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DISEASES OF THE STOMACH AND INTESTINES

Diseases of the Stomach and Intestines. By Robert Coleman Kemp, M.D., Professor of Gastro-intestinal Diseases, New York School of Clinical Medicine. Octavo of 766 pages, with 279 illustrations. Philadelphia and London: W. B. Saunders Company, 1910. $6.00 net; Half Morocco. $7.50 net.

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1910

DISEASES

OF THE

Stomach and Intestines

BY

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WITH 280 ILLUSTRATIONS, SOME IN COLORS

PHILADELPHIA AND LONDON
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1910

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TO

William H. Thomson, M. D., LL. D.,

CONSULTING PHYSICIAN TO THE ROOSEVELT AND RED
CROSS HOSPITALS AND LATE PRESIDENT
OF THE NEW YORK ACADEMY
OF MEDICINE,
THIS VOLUME IS INSCRIBED
AS A TRIBUTE TO HIS HIGH PROFESSIONAL ATTAINMENTS AND IN REMEMBRANCE OF HIS
MANY ACTS OF KINDNESS,
BY THE AUTHOR

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PREFACE

In view of the excellent works on diseases of the stomach and intestines that have been placed before the medical profession, the publication of a new book on these subjects might almost seem to be superfluous. From a great accumulation of material, it is often difficult for the general practitioner to select simple and practical methods, and it is the endeavor that this volume should render service in this special direction.

Many physicians have neither time nor opportunity to devote to a practical clinical course, and next in value to this for the purpose of instruction is the employment of photography to demonstrate the methods of diagnosis and treatment. Of this I have endeavored to take advantage. Many of the illustrations are from photographs of patients at the Manhattan State Hospital, taken by Mr. Hill, the official photographer, for whose services I am indebted to the courtesy of Dr. Wm. Mabon, the Medical Superintendent. Some of the illustrations have been made by my artist from models.

As visceral displacements have recently assumed an important position, their symptoms, diagnosis, and treatment, notably by mechanical methods, are specially described. Typhoid fever is included in this volume on account of its intestinal complications and for the purpose of differential diagnosis.

A chapter is devoted to *Diverticulitis*, which has become an important subject.

The endeavor has been made to indicate as clearly as possible the conditions which call for surgical procedure.

ROBERT COLEMAN KEMP.

NEW YORK CITY,
February, 1910.

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DISEASES

OF THE

STOMACH AND INTESTINES

PART I

CHAPTER I

ANATOMY OF THE STOMACH AND INTESTINES

ANATOMY OF THE STOMACH

A BRIEF description will be given of the anatomy of the stomach, but for a complete exposition of the subject the reader is referred to any standard anatomy.

The stomach lies in the epigastric and left hypochondriac regions, about five-sixths of it to the left of the median line. The larger end, the fundus, fits into the concave left vault of the diaphragm. It is a pyriform sac, with longitudinal diameter slightly oblique, and lying transversely across the abdomen (Fig. 1).

The cardiac orifice (*C*), the junction of the esophagus and stomach (the esophageal orifice), is fixed and lies behind or a little to the left of the sternal junction of the left seventh cartilage (seventh rib), or about 1¼ inches from the edge of the sternum, in the left parasternal line, on a level with the spinous process of the ninth dorsal vertebra. The cardia is situated 4½ inches from the anterior surface of the abdomen. The point of communication with the small intestine is called the pylorus (*P*), and shows a furrow on the outer surface and within a protruding fold (the valve of the pylorus).

The pylorus (*P*) lies between the right sternal and parasternal lines, slightly below the tip of the ensiform process, and corresponds to the spinous process of the twelfth dorsal vertebra. It descends slightly when the stomach is distended and moves somewhat to the right. A line (+), drawn in the axis of the esophagus through the stomach to its lower border, cuts off about one-fourth of the organ to the left. This portion is called the greater cul-de-sac or fundus (*F*).

The fundus (*F*) rises as high as the lower border of the left fifth rib in the mammillary line, slightly above and behind the apex of the heart, and from 1 to 2 inches higher than the cardia. It is in contact with the diaphragm above, and to the left with the spleen and left kidney.

The lesser curvature (*L*) lies to the left of the vertebral colum passes downward and parallel with it, and then crosses it to t upper border of the pylorus.

The greater curvature (*G*) forms the fundus and lower border the stomach and extends to the lower border of the pylorus. T lower border, when the organ is distended, lies about two to thr fingers' breadth (1½ to 2¼ inches) above the umbilicus.

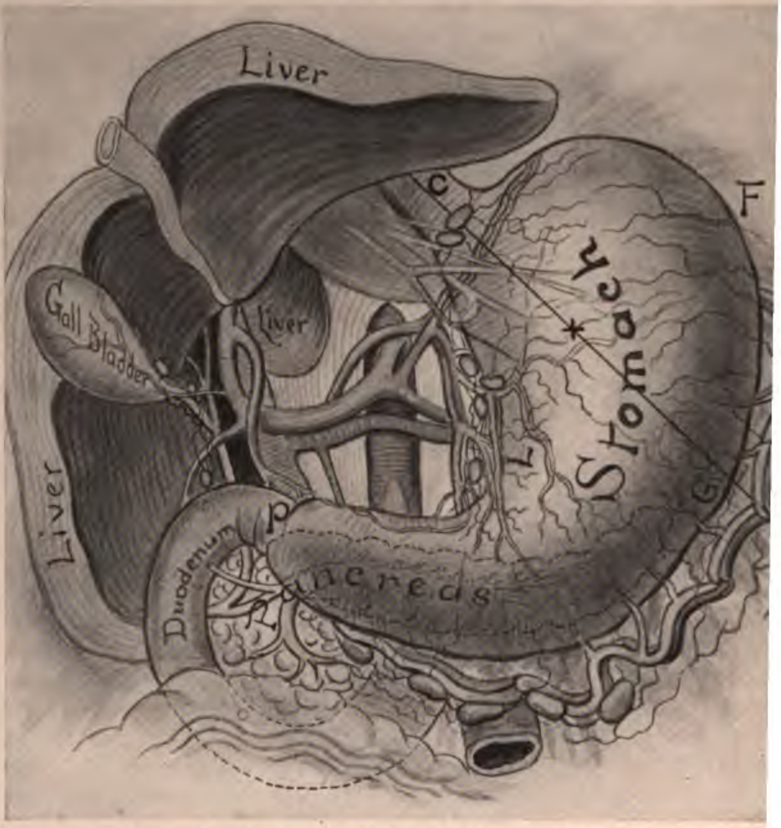

Fig. 1.—The stomach: *C*, Cardia; *F*, fundus; *P*, pylorus; *L*, lesser curvat *G*, greater curvature (modified from W. J. Mayo).

The volume of the stomach varies according to its conte Dehio has shown that the healthy stomach when empty is contrac and hidden away in the left cavity of the diaphragm, and it is colon that we then demonstrate by percussion.

The pancreas and splenic vessels lie behind the stomach. anterior surface is overlapped above by the liver, the left lung, the seventh, eighth, and ninth ribs. Below it is in relation with abdominal wall.

The pyloric end, the lesser curvature, and the cardia lie behind and beneath the quadrate and left lobes of the liver.

Traube's space is the area in which the stomach lies in direct contact with the ribs, and is bounded above by the liver and left lung, externally by the spleen, and the inner border is formed by the free costal margin. Both here and in the epigastric region pure gastric tympany can be elicited.

When the stomach is distended, the lesser curvature is directed obliquely backward toward the spine, the posterior wall looking somewhat downward and the anterior wall slightly upward. The transverse colon, if distended, may overlap the greater curvature, and the latter tends to fall away from the abdominal wall when the patient is in the dorsal position. The transverse colon lies ordinarily below the greater curvature. With moderate distention, the average length from fundus to pylorus is 10 to 12 inches; from the lesser to greater curvature, 4 to 5 inches; from the anterior to the posterior wall, about 3 to 3½ inches.

The average capacity is variable. It may contain even as much as 2 quarts. A plane drawn transversely through the base of the lesser curvature will lie parallel with the plane of the diaphragm.

The lesser omentum extends from the lesser curvature to the liver above, and the great omentum is suspended from the greater curvature, protecting the viscera.

The blood-vessels enter the upper and lower borders, and thus divide the surface into two equal parts. They mark the greater and lesser curvatures.

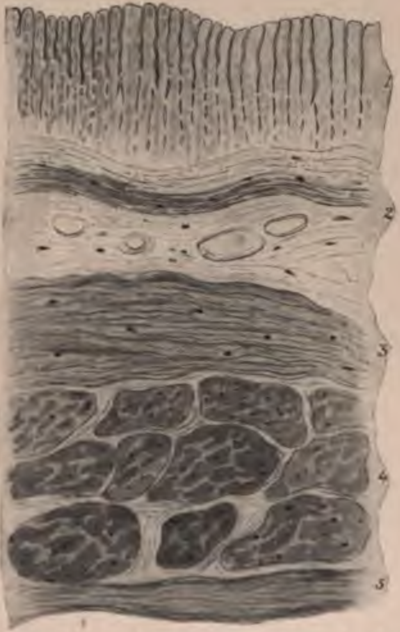

Fig. 2.—Vertical section of the stomach: 1, Mucosa; 2, submucosa; 3, 4, muscularis; 5, serosa.

Structure of the Stomach.—The stomach consists of four coats: serous or peritoneal, muscular, submucous or areolar, and mucous. The peritoneal coat forms a thin, transparent, elastic membrane, and closely covers the organ except along the curvatures, where it is more loosely attached for the passage of the blood-vessels.

The muscular coat consists of three sets of fibers (Fig. 2), disposed in layers, the outer or longitudinal, middle or circular, and inner or oblique. The last is a continuation of the circular fibers of the esophagus and the fibers descend obliquely from the cardia upon the anterior and posterior surface, and, spreading out like fan, terminate at the greater curvature.

The submucous coat consists of areolar tissue, connecting t mucous and muscular coats. The blood-vessels subdivide there

The mucous membrane is soft, smooth, somewhat pulpy, a of pink color, thickest in the pyloric region and thinnest at t fundus. It constitutes the glandular layer of the organ. It covered by columnar epithelial cells, which extend for a varia distance into the mouths of the glands. There are about five mill glands in the stomach, tubular in form, and perpendicular to surface. They are surrounded by fibrous tissue and lymphoid c and by a thin muscle layer (muscularis mucosæ).

The glands are composed of a mouth, neck, body, and base; a several tubules, from two to even four or five, may end in one mou On microscopic examination, the dots appearing on the surface the mucosa are the openings of the glands. There are three varie of glands in the stomach:

Cardiac or fundus glands. Pyloric glands. Mucous glands.

Cardiac or *fundus glands* are the most numerous. They fill greater part of the stomach and are characterized by the short of their mouths and the length of the glands. They contain varieties of cells. Cells bordering on the lumen of the tube, w are small, granular, and polyhedral or columnar, the chief or princ cells, and which only stain to a slight extent with anilin dyes. other cells, parietal or oxyntic, lie between the principal cells the membrana propria (Fig. 3). They are most numerous in necks of the glands, larger than the chief cells, are oval or ang and finely granular in structure. They have strong affinity anilin dyes.

Pyloric Glands.—These are characterized by the greater le of their mouths, which are lined by cylindric epithelium. They found only in the region of the pylorus. The body or secre portion of the gland is represented by a single layer of short finely granular columnar cells, resembling the chief cells of the fu glands. There are also a few isolated cells (Nussbaum) w resemble, in structure and in their behavior to anilin dyes, the par cells of the fundus glands (Fig. 4).

Besides these specific glands, a number of *mucous glands* found near the pylorus.

The hydrochloric acid is secreted by the parietal cells; p and the milk-curdling ferment by the principal cells of the fu and pyloric glands. Some consider the mucus to be also a pr of the cylindric goblet-cells lining the stomach and the wider por of the glandular ducts. The ferments do not exist as such i cells, but as zymogens, which are transformed into ferments thr the activity of the free hydrochloric acid.

Blood-vessels, Lymphatics, and Nerves of the Stomach.–

arteries of the stomach are derived from branches of the celiac axis; the gastric and pyloric branch of the hepatic artery supplying the upper curvature and forming the superior ventricular arch and the right gastro-epiploic from the hepatic, and the left gastro-epiploic and vasa brevia from the splenic form the inferior ventricular arch.

They reach the stomach between the folds of peritoneum and ramify between the muscular coats, giving off a number of capillaries and dividing into small vessels in the submucosa, and finally enter the mucous membrane, and pass between the tubuli, where they form a plexus of fine capillaries both on the walls of the tubules and around the mouths of the glands.

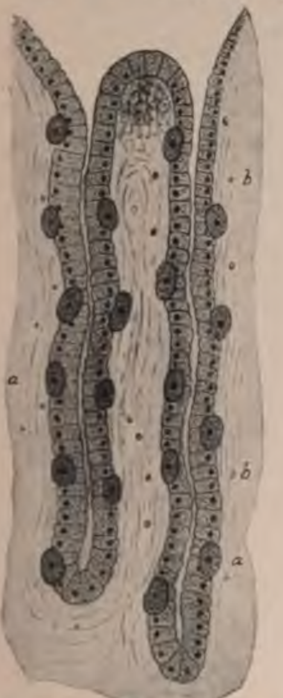

Fig. 3.—Cardiac gland: *a*, Parietal cells; *b*, principal cells.

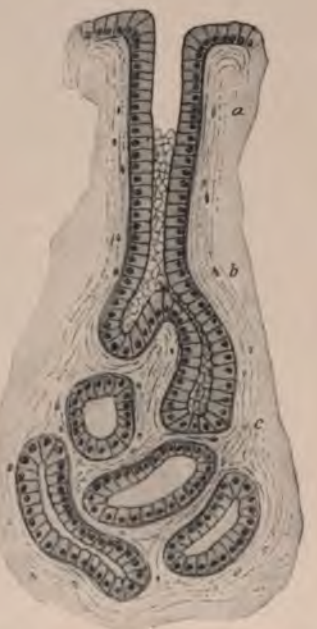

Fig. 4.—Pyloric gland: *a*, Mouth; *b*, neck; *c*, fundus.

The *veins* arise from this capillary network and pass nearly straight through the mucous membrane between the glands. They pierce the muscularis mucosæ and form a plexus in the submucosa, and finally form the coronary and pyloric veins emptying into the portal vein, the right gastro-epiploic vein emptying into the superior mesenteric vein, and the left gastro-epiploic vein emptying into the splenic vein.

The *lymphatics* extend directly to the surface of the mucosa. They form a dense network of lacunar spaces between and among the gland tubuli, which they enclose, as well as the blood-vessel with sinus-like dilatations. The lymph is collected near the surfa of the mucous membrane into vessels which form loops and posse dilated extremities. They are less superficial than the capillarie though the lacunar spaces extend as far as the basement-membra of the surface. The lymphatic glands extend *along the lesser curv ture to the cardia,* while they are present on the greater curvatu from the *pylorus to only about one-fourth to one-third of the distan* This fact is of important consideration in gastrectomy for carcinom as the lesser curvature must, therefore, be entirely removed.

Nerves.—The left vagus supplies the anterior surface of t stomach. The right vagus supplies the posterior surface with on one-third of its fibers, the remainder passing to the other viscera.

Branches of the sympathetic nerves pass from the celiac plex and anastomose with the vagi. These nerves with numerous gang form a network in the submucosa.

ANATOMY OF THE INTESTINES

The intestinal canal is divided into two parts: the small intesti and the large intestine; the former about 7.6 meters (25 feet) lo and the latter 1.5 to 1.8 meters (5–6 feet) long.

The small intestine is subdivided into three portions: the du denum, jejunum, and ileum, and lies, excepting the duodenum, the inner side of the large intestine, and is connected to the poster abdominal wall by the mesentery, which last encloses the jejun and ileum throughout.

The Duodenum.—The duodenum, which is about 10 to 12 inc (25.5–30.5 cm.) long, is not suspended by the mesentery and is most fixed and widest part of the small intestine, having a diame of 1½ to 2 inches (3.81–5.08 cm.). It is curved like a horsesh surrounds the pancreas, and is divided into four parts (Fig. 5).

The superior horizontal part of the duodenum is about 2 inc (5.08 cm.) long, begins at the pylorus at the level of the first lum vertebra, and passes slightly upward and to the right of the g bladder. It is the most movable portion, is surrounded by peritoneum, and suspended chiefly by ligaments from the hilus the liver and neck of the gall-bladder. The quadrate lobe and n of the gall-bladder lie above it; below it is the pancreas, and beh it the common bile-duct and hepatic vessels.

The descending portion of the duodenum, about 3 inches (7.5 c long, commences at the neck of the gall-bladder and runs vertic to the third or fourth lumbar vertebra on the right side and touc the right kidney. The transverse colon passes in front of it; on left side is the pancreas, and the common bile-duct lies a little po rior. At its inner and back part, about 4 inches from the pylorus, common bile-duct and pancreatic duct enter it and form the diverti lum or ampulla of Vater.

The third or transverse portion of the duodenum, about 5 inches (12.5 cm.) long, extends from the right side of the body of the third or fourth lumbar vertebra across the spine, and slightly ascends to the left side of the spine. The superior mesenteric vessels cross it, as does the mesentery. The lower layer of transverse mesocolon lies in front. The pancreas and superior mesenteric artery lie above, and the aorta, vena cava, and crura of the diaphragm behind it. It is the most fixed portion of the duodenum.

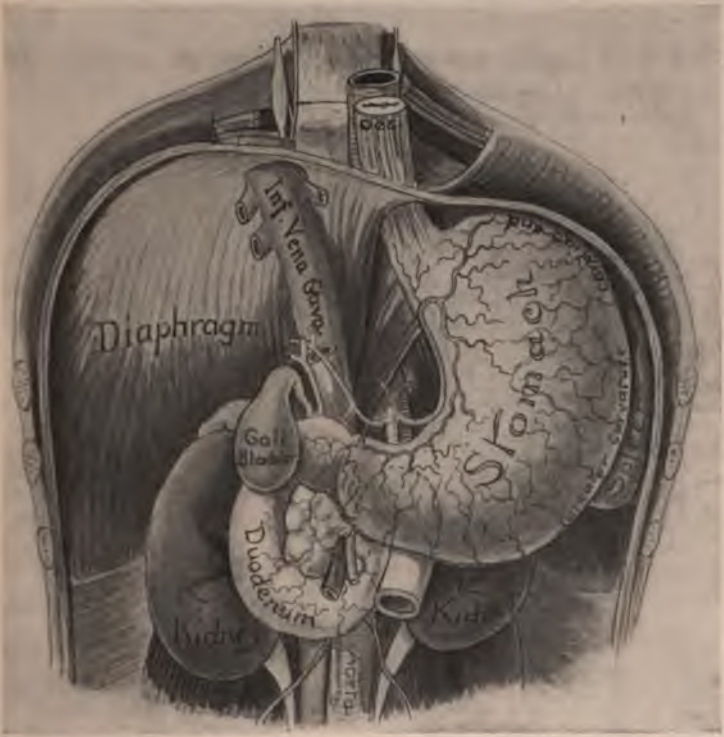

Fig. 5.—Stomach and duodenum, liver and intestines removed, and showing anatomic relations of the duodenum (after Testut).

The fourth or ascending portion of the duodenum, about 1 to 2 inches (2.54–5.08 cm.) long, ascends vertically along the left side of the spine from the third or fourth lumbar vertebra to the side of the second or first lumbar vertebra. It is firmly fixed by the suspensory muscle of the duodenum (muscle of Treitz), which descends from the left crus of the diaphragm. Anteriorly are the transverse colon and transverse mesocolon (lower layer). It terminates in the jejunum (usually opposite the second lumbar vertebra) and forms the duodenojejunal flexure.

Jejunum and Ileum.—They form the continuation of the duodenum. It is hard to determine where the one ends and th other begins. The jejunum occupies the upper two-fifths of th remaining small intestines, or about 9 feet, 7 inches (2.9 meters) the ileum the lower three-fifths, or about 14 feet, 5 inches (4.3 meters and ends at the ileocecal junction. Their position is not fixed, bu the jejunum is more apt to occupy the left side of the abdomina cavity, with the loops more transverse; while the ileum is usuall found on the right side and in the pelvis, with the loops more vertica

The coils of the jejunum and ileum are very movable and ar completely invested by peritoneum. They are supported an attached to the posterior parietes by the mesentery, which is attache above to the left of the vertebræ on a level with the lower border o the pancreas. The mesentery runs downward and to the right an presents the form of a fan, the intestines hanging on it in the for of coils.

The diameter of the jejunum is about 1½ inches (3.81 cm.), an this gradually diminishes in size to the ileum; and in this, in tur the diameter decreases until its entrance into the large intestin The ileum passes nearly perpendicularly into the ascending colo its mucosa forming a double valve (valvula Bauhini).

The jejunum and ileum are the most movable parts of the inte tinal tract. They are often met with in hernias, and if pregnanc a tumor, or ascites are present, the intestines move up and escap compression.

Occasionally Meckel's diverticulum (the remains of the vitellin duct), a process 2 or 3 inches long, is given off from the ileum, on a average of 1 to 2 feet above the ileocecal junction. It is of impor ance in reference to intestinal obstruction.

Arterial Supply of Small Intestine.—The duodenum i supplied by the pyloric branch of the hepatic, by the superior pa creaticoduodenal branch of the gastroduodenal branch of the hepati and by the inferior pancreaticoduodenal branch of the superi mesenteric, and the jejunum and ileum by the superior mesenteri artery. They branch into small vessels which run through th intestinal wall, ramify in the submucosa, and form the capillar system of the villi and glands.

Veins.—The venous blood flows partly into the superior gastr vein and partly into the superior mesenteric vein and empties in the vena porta.

Lymphatics.—The lymphatics are divided into those of t mucous membrane and muscular coat, and form plexuses. Th run between the folds of the mesentery and end in the mesenter lacteals, and so on into the intestinal lymphatic trunk and thorac duct. They are provided with valves to prevent a backward flo

Nerves.—The duodenum is supplied by the hepatic plexus, branch of the celiac plexus, with branches of the right vagus.

The superior mesenteric plexus, formed by nerves from t celiac plexus, the semilunar ganglia, and right vagus, supply the jejunum and ileum.

The nerves enter the intestinal wall with the blood-vessels and form a subserous net. They then penetrate the longitudinal muscular fibers and form between these and the circular muscular fibers ramifications consisting of numerous groups of multipolar cells (Auerbach's plexus), from which fine branches supply the muscular tissue. Other branches penetrate the circular muscular layer to the submucosa, where they form the submucous nerve plexus (Meissner's plexus), and branches supply the muscularis mucosæ, the muscles of the villi, and end in the mucosa.

Structure of the Small Intestine.—The small intestine is composed of four coats: serous or peritoneal, muscular, submucous, and mucous (Fig. 6).

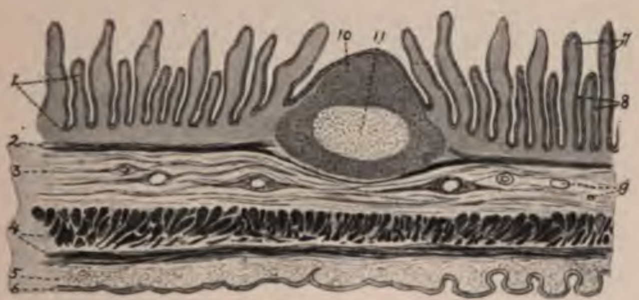

Fig. 6.—Longitudinal cross-section through the wall of the small intestine: 1, Mucous layer; 2, muscularis mucosæ; 3, submucous layer; 4, muscular layer; 5, subserosa; 6, serous layer; 7, intestinal villi; 8, intestinal glands (Lieberkühn); 9, blood-vessels; 10, solitary lymph nodule; 11, center of same.

The serous coat is formed by the visceral layer of the peritoneum. The muscular coat consists of an internal circular layer and an external longitudinal layer, the former being considerably the thicker.

They consist of bundles of unstriped muscular tissue supported by connective-tissue fibers. The submucosa consists of connective tissue in which blood-vessels, lymphatics, and nerves ramify.

The mucous membrane comprises a thin muscular layer (muscularis mucosæ), containing circular and longitudinal fibers and the tunica propria of the mucous membrane, which is made up principally of reticular connective tissue, with leukocytes, glands, villi, and an epithelial covering.

The mucosa is of a grayish-red color, appears velvety, and forms crescentic folds, set transversely to the long axis of the intestine (valvulæ conniventes of Kerkring, Fig. 7). Each valve extends from one-half to two-thirds of the circumference of the gut, and they may be 2 inches long and ⅓ inch wide. They begin a short distance below the pylorus, at the middle of the jejunum commence to diminish in size, and gradually disappear at the lower part of the ileum. They serve to increase the absorptive surface of the mucous membrane.

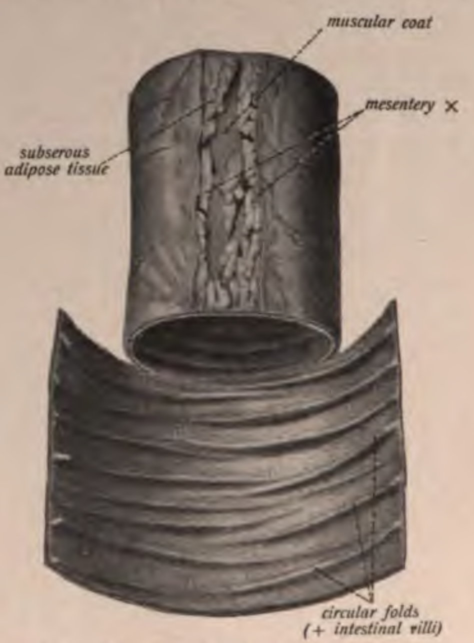

Fig. 7.—A portion of the jejunum showing the circular folds or valvulæ conniventes of Kerkring (Sobotta).

Microscopic Anatomy of the Small Intestine.—The inner surface of the small intestine is composed of villi and glands, the surface being covered by a layer of columnar epithelial cells, with striated borders and some goblet-cells.

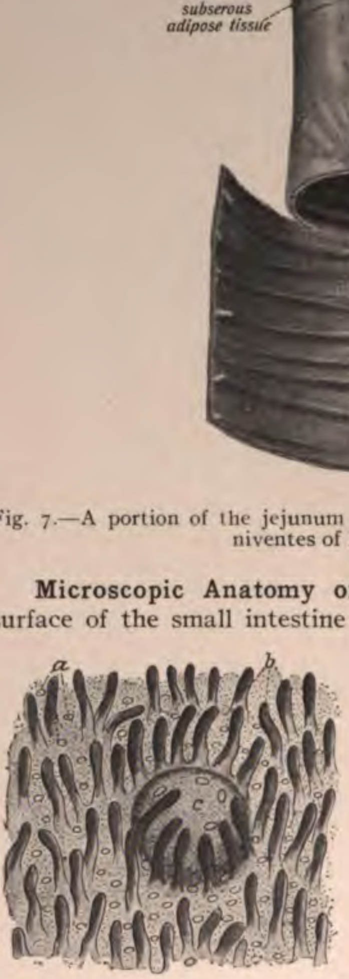

Fig. 8.—Mucous membrane of ileum; *a*, Intestinal glands (Lieberkühn); *b*, intestinal villi; *c*, solitary lymph nodule (follicle).

The villi are formed chiefly by elevations of the tunica propria of the mucous membrane. They are from .5 to .7 mm. in height and about .1 to .2 mm. in width, and number about ten millions (Fig. 8).

Each villus has a central space for chyle, which cavity is covered with endothelial cells and connects with the lymphatics of the mucosa. The villus has blood-vessels and muscular fibers which are derived from the muscularis mucosæ. It expands when filling up with blood, and when the muscle contracts it shrinks.

The villi thus have an action of suction and pumping, and also form the chief organs for absorption in the small intestine. Around the villi are numerous glands; the tubular glands of Lieberkühn and the acinous glands of Brunner. The latter are confined to the duodenum.

The glands of Lieberkühn resemble in structure the tubular glands of the stomach, and cover almost the entire surface of the small and large intestine. Each tubule is from .3 to .4 mm. long, and opens without ramifications. They form the chief organ of intestinal secretion and number over forty millions.

Brunner's glands are found only in the duodenum and are most abundant at its commencement, diminishing in number lower down. They lie beneath the mucosa, being embedded in the submucosa. They resemble the pyloric glands of the stomach, but are more branched and convoluted, and their ducts are longer. They are lined with columnar epithelium. The duct of the gland passes through the muscularis mucosæ and opens on the surface of the mucosa (Fig. 9).

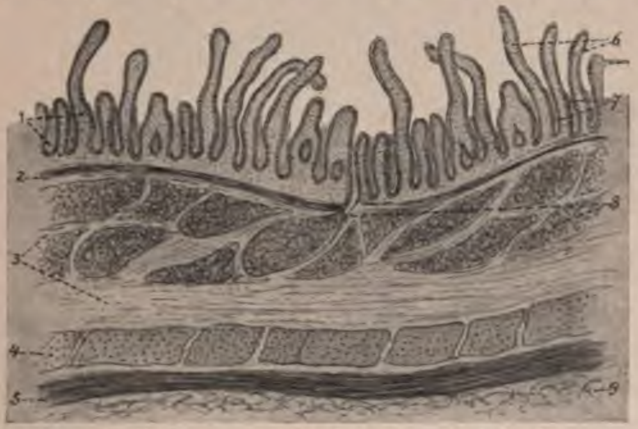

Fig. 9.—Longitudinal cross-section through wall of duodenum: 1, Mucous layer; 2, muscularis mucosæ; 3, submucous layer; 4, circular muscular layer; 5, longitudinal muscular layer; 6, intestinal villi; 7, intestinal glands (Lieberkühn); 8, Brunner's duodenal glands; 9, serous layer.

Solitary follicles (or glands) are scattered throughout the mucous membrane of the small intestine and are most numerous in the lower ileum. They have a diameter of 2 to 6 mm. (Fig. 10).

The follicles consist of a dense retiform tissue packed with lymph-corpuscles and permeated by capillaries. They have no ducts. The spaces in this tissue are continuous with lymph-spaces at the base of the gland and the base of the follicle is in the submucous tissue. The gland enters the mucous membrane, causing a slight projection of its epithelial layer. Lymph-cells develop in these follicles. There are no villi on their surface.

These follicles are scattered singly through the intestine as solitary glands, or collected into groups, known as Peyer's patches or

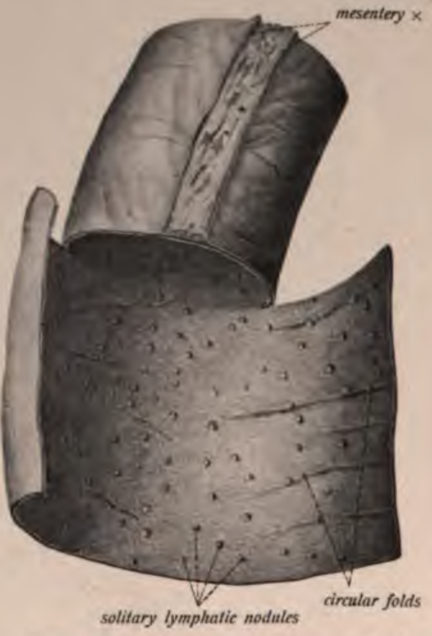

Fig. 10.—A portion of the ileum showing solitary lymphatic nodes (Sobotta).

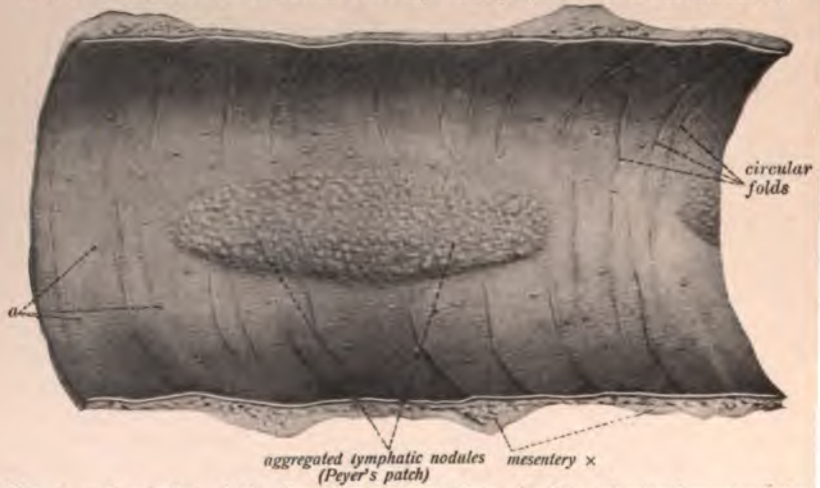

Fig. 11.—A portion of the ileum, cut open along the line of attachment of the mesentery, showing Peyer's patch and solitary lymphatic nodes (*a*) (Sobotta).

plaques, or as the agminate glands. These last may be from 1 to 3 inches long and ½ inch wide, usually oval, with the long axis parallel with that of the intestine. They lie generally opposite the attachment of the mesentery, are twenty to thirty in number, and are found chiefly in the ileum, though a few are present in the jejunum (Fig. 11).

Anatomy of the Large Intestine.—The large intestine, which is about 5 to 6 feet (1.5–1.8 meters) long, extends from the termination of the ileum to the anus, and is divided into the cecum (or caput coli), the colon, and the rectum. Its caliber is largest at the cecum, and this gradually decreases until it reaches the ampulla of the rectum, when it again increases in size.

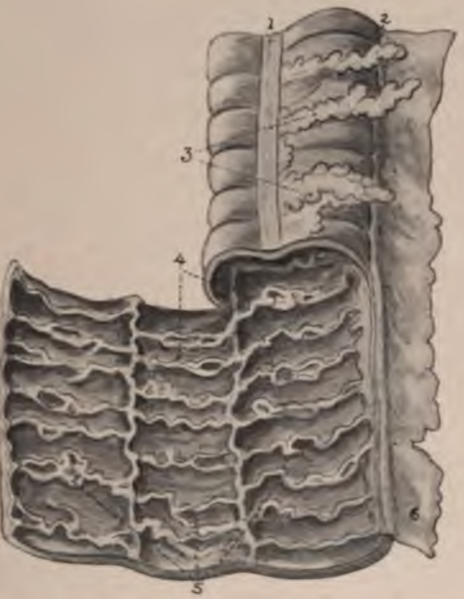

Fig. 12.—Partial section of colon: 1, Free tenia; 2, tenia mesocolica; 3, appendices epiploicæ; 4, mucosa; 5, semilunar folds; 6, mesocolon.

The large intestine, excepting the rectum, is characterized by three longitudinal unstriated muscular bands or teniæ (Fig. 12), with sacculation of the walls between these bands and by the appendices epiploicæ, or external pouches formed by the peritoneal covering and containing fat.

The circular muscular fibers also accumulate in bands with intervals between them, thus forming expansions or semilunar folds across the colon (haustra coli) (Fig. 13).

The cecum (caput coli) is that part of the colon lying below the ileocecal valve (Fig. 14). It is about 3 inches (7.5 cm.) broad and

2½ inches (6.3 cm) long, and lies in the right iliac fossa above the outer half of Poupart's ligament, being completely covered by peritoneum. When filled, it is situated close to the abdominal wall. The vermiform appendix, a small blind tube, hollow nearly to the tip, is given off generally from the posterior and inner portion of the caput coli, about $\frac{11}{16}$ inch (1.7 cm.) below the ileocecal valve.

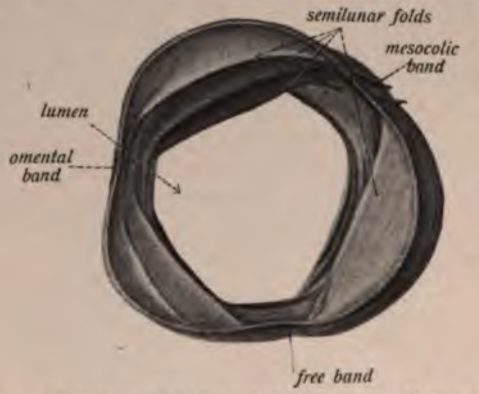

Fig. 13.—A segment of the colon (Sobotta); Haustra coli.

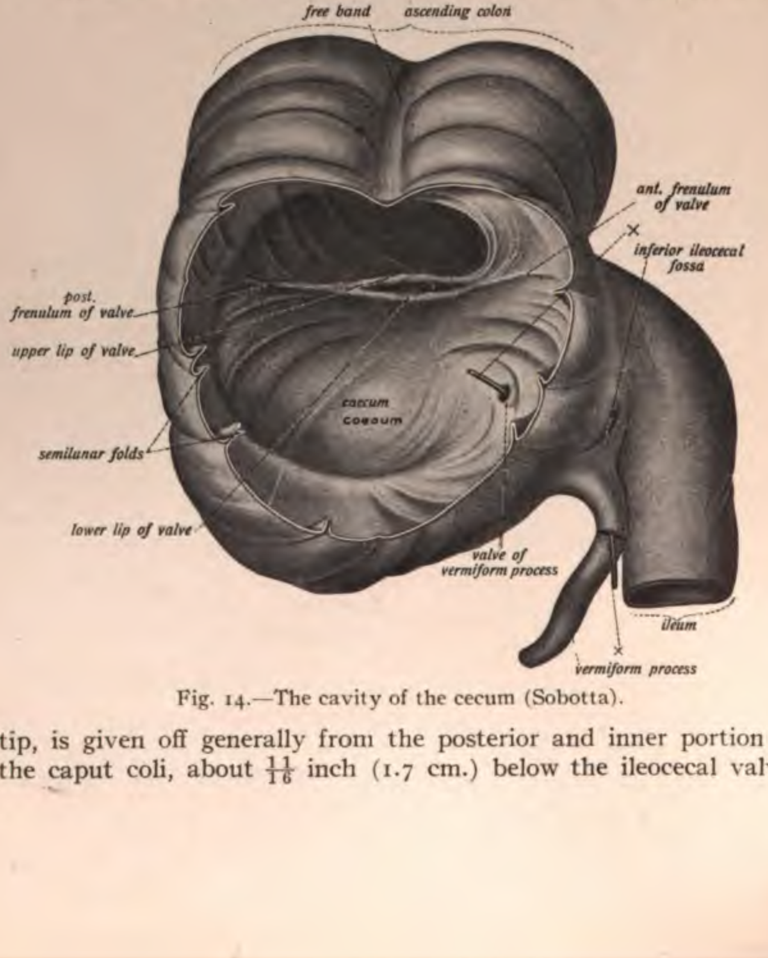

Fig. 14.—The cavity of the cecum (Sobotta).

Its average length is 3¾ inches (9.2 cm.). It may be much shorter or longer. The diameter is about ¼ inch (6 mm.) at base and $\frac{3}{16}$ inch (5 mm.) at apex. It may be slightly larger at the middle. It is usually guarded by a valve (crescentic fold) where it enters the cecum. It has a meso-appendix (mesentery) only extending about two-thirds its length. The position of the appendix is not fixed and it may point in various directions. The colon is depicted in Fig. 21.

The ascending colon, about 8 inches (20 cm.) in length, extends vertically upward from the cecum to the inferior surface of the right lobe of the liver to the right of the gall-bladder, at which point it bends to the left (hepatic flexure). It passes along the posterior abdominal muscles and lower part of the right kidney, and is in relation to the abdominal wall in front. It is bound posteriorly by connective tissue to the muscles, and is only covered by peritoneum anteriorly and laterally.

The transverse colon, with an average length of 20 inches (51 cm.), extends from the hepatic flexure beneath the liver transversely across the abdominal wall, with a slightly downward and forward convexity at its center, to the spleen in the left hypochondrium (splenic flexure). It has a long mesentery, transverse mesocolon, connecting it with the posterior abdominal wall, and is the most movable part of the large intestine. It usually corresponds to a line separating the umbilical and epigastric regions. The liver, gall-bladder, greater curvature of the stomach, and lower end of the spleen lie above it; the small intestine lies below; the descending duodenum and small intestine behind; the great omentum and abdominal wall in front.

At the splenic flexure below the lower end of the spleen, the colon turns downward (descending colon). This is about 8½ inches (21.5 cm.) long, and extends from the splenic flexure vertically through the left hypochondriac and lumbar regions to the sigmoid flexure. It is covered anteriorly and laterally by the peritoneum, and passes down in front of the left kidney and quadratus lumborum and iliac muscles, to the left iliac fossa into the sigmoid flexure.

The sigmoid flexure of the colon (S. romanum) is an S-shaped curve, about 13 inches (31 cm.) long, beginning at the iliac crest and ending at the brim of the true pelvis opposite the left sacro-iliac articulation. The upper, or colic, limb tends down, inward, and forward toward Poupart's ligament, while the lower, or rectal, limb hangs down into the true pelvis, where it joins the rectum. The sigmoid flexure has a complete peritoneal covering, or mesentery, is very movable, and is the narrowest portion of the large intestine.

The rectum, which is about 8 to 9 inches (20–23 cm.) long, passes from the left sacro-iliac junction obliquely to the middle of the sacrum, and follows it down to the bottom of the pelvis to about 1 inch (2.5 cm.) below the tip of the coccyx, where it passes downward and backward to end in the anus. It is divided into three portions.

Only the first part, 3½ inches (9 cm.), of the rectum is completely invested with peritoneum (mesorectum), and it is attached to the sacral vertebræ. The second part is partially invested with peritoneum (pouch of Douglas), which lies anterior and ascends over the bladder or vagina. The third part has no peritoneal investment.

The lower half of the rectum passes between the organs occupying the pelvic floor and is adherent to them by connective tissue. The rectum is surrounded by connective tissue below the pouch of Douglas. It is widest at the point opposite the prostate, there forming the ampulla of the rectum.

The outer longitudinal muscular fibers of the rectum are not arranged in teniæ, as in the colon, but are present in all parts of its circumference. The inner circular layer of muscle-fibers increases in density from above downward and forms a thick ring at the anal opening (the internal sphincter). The walls of the rectum are connected at the anus with the sphincter ani and levator ani muscles, which are of importance in defecation.

Arterial Supply of the Cecum and Colon.—They are the ileocolic, colica dextra, and colica media from the superior mesenteric artery; the colica sinistra and sigmoid from the inferior mesenteric artery.

Veins of the Cecum and Colon.—These are the superior and inferior mesenteric, emptying into the portal system.

Lymphatics of the Cecum and Colon.—Those of the sigmoid colon empty into the lumbar glands; those of the rest of the colon into the mesenteric glands.

Nerves of the Cecum and Colon.—The cecum, ascending colon and right half of the transverse colon are supplied by the superior mesenteric plexus, a branch of the celiac plexus. The rest of the colon, including the sigmoid flexure, is supplied by the inferior mesenteric plexus, a branch of the aortic plexus.

Arterial Supply of the Rectum.—They are the superior hemorrhoidal (of the inferior mesenteric); the middle hemorrhoidal (of the internal iliac); the inferior hemorrhoidal (of the internal pudic) branches from the sacromedia (of the abdominal aorta); branches from the sciatic (of the internal iliac); in the female, branches from the vaginal.

Veins of the Rectum.—They are chiefly from the superior hemorrhoidal, passing to the inferior mesenteric and to the porta system; part of the other hemorrhoidal veins empty into the interna iliac veins, and so enter the general venous system.

Anastomosis thus occurs in the rectum between the portal and the general venous system, and there is a communication also with the other abdominal veins.

Lymphatics of the Rectum.—They empty into the sacral and lumbar glands from the rectum; from the anus into the inguina glands.

Nerves of the Rectum.—The cerebrospinal nerves come from the sacral plexus; the sympathetic nerves from the inferior mesenteric and superior hypogastric plexus.

HISTOLOGY OF THE LARGE INTESTINE

The large intestine (colon), like the small intestine, consists of four coats: serous, muscular, submucous, and mucous.

The longitudinal muscular fibers of the large intestine, however, are arranged in three bands (teniæ) running along the wall, as already described.

The serosa and submucosa resemble those of the small intestine in structure.

The mucosa of the large intestine differs from that of the small intestine in that the villi and circular folds of Kerkring (valvulæ conniventes) are absent. The glands of Lieberkühn are somewhat longer and at times curved.

The mucous membrane of the rectum is thicker, redder, and more generously supplied with blood-vessels than that of the colon.

When the rectum is empty the mucous membrane of the upper part is thrown into a multitude of superficial transverse velvety folds. From two to seven folds (Houston's valves) are made more prominent by distention (Gant).

Just above the anus are a number of longitudinal folds (columns of Morgagni), extending for $\frac{1}{3}$ to $\frac{3}{5}$ inch (8.46–15.23 mm.).

The mucous membrane of the rectum consists of columnar epithelium, except at the lowest portion, a narrow layer of stratified pavement-like epithelium, transitional between skin and rectal mucosa. The upper part of the rectum resembles the colon.

CHAPTER II

PHYSIOLOGY OF DIGESTION

THE stomach and intestines form an important part of the digestive tract, and in order to understand their functions it will be necessary to review briefly the process of digestion. This term includes those processes which convert the food into such condition that it becomes fit to enter the circulation and afford nutrition to the human organism. These changes are brought about by means of certain ferments contained in the saliva, gastric juice, bile, pancreatic juice and intestinal juice, which are a part of the human organism at birth.

There are certain fermentative and putrefactive processes which take place in the gastro-intestinal canal, the result of bacterial invasion, which play an important part in the physiology and pathology of this tract.

The first ferment (ptyalin) with which the food comes in contact by the act of chewing, is found in the saliva. The latter is alkaline in reaction, of low specific gravity (1.002 to 1.0009), and contains water, ptyalin, mucus, epithelia, albumin, and salts.

The ptyalin, which converts starch into maltose, or sugar, begins its action on the food already in the mouth, but the principal work is done during the first period of digestion within the stomach. Under normal conditions during this early stage of gastric digestion the free hydrochloric acid is becoming combined acid, and the action of the ptyalin continues; but if the free hydrochloric acid be excessively secreted, further digestion of the starch is interfered with; this physiologic fact has a bearing on the treatment of hyperchlorhydria. We must remember that thorough mastication of the food, which promotes salivary secretion, and the care of the teeth have an important bearing on digestion. It has been demonstrated that acid fermentation in the mouth interferes with the action of the saliva, and that cleansing the mouths of nursing infants will diminish fermentative processes in the gastro-intestinal tract.

Hemmeter has apparently recently demonstrated on dogs that the salivary glands secrete a chemical substance during mastication which *passes into the blood* and starts up the secretion of the gastric juice. After extirpation of the glands, HCl and rennin were much reduced. Intravenous injection of salivary gland extracts partially restored gastric secretion.

THE GASTRIC JUICE

Hydrochloric acid was discovered in the gastric juice by Prout in 1824; and Beaumont, in 1833, by his experiments on St. Martin with his gastric fistula, greatly advanced our knowledge. Schwam, in 1836, discovered the pepsin ferment; and Bedder and Schmidt, in 1854, showed that the acid of the gastric juice is hydrochloric acid.

The gastric juice is a clear, colorless fluid, of an acid reaction and a specific gravity of 1.002 to 1.003. It contains water, salts, inorganic matter, proteids, hydrochloric acid, pepsin, rennet, and recently a fat-splitting ferment has been discovered.

The quantity of this secretion in twenty-four hours is estimated to be about 3 pints. The degree of acidity varies from 0.1 to 0.2 per cent. Pepsin and rennet when first secreted are inactive bodies, known as pepsinogen and rennet-zymogen, but on coming into contact with the hydrochloric acid become converted into active pepsin and rennet. Various theories have been advanced to explain the production of the gastric juice, how an inorganic acid comes to be secreted by the blood which is of alkaline reaction. We are so far forced to accept the view that the secretion of the gastric juice must be due to the specific action of the cells.

Through the combined action of hydrochloric acid and pepsin, it converts the albuminates into propeptones and peptones, which are more soluble. The rennet ferment curdles the milk. A small percentage of fat is split into fatty acids.

It has the property of converting cane-sugar into grape-sugar, and gelatin into a soluble peptone which does not coagulate.

Some of the substances contained in the liquefied chyme are absorbed through the stomach-walls, such as peptone, sugar, salts, and possibly propeptone. Meltzer has demonstrated that only a small quantity of water is absorbed in the stomach. The residue of the gastric contents passes into the small intestine, where further digestive processes occur through the action of the other ferments and the principal absorption takes place.

The motor function of the stomach is of importance in the process of digestion, as by its active and passive movements physical changes are brought about in the ingesta. The food is brought more closely in contact with the stomach-walls and becomes more liquefied. The pylorus opens and closes at intervals, and allows the entrance of the chyme into the small intestine, the exact intervals of which we do not know. About two to three hours after a small meal and six to seven hours after a large meal the stomach is empty.

Cannon[1] has studied the movements of the stomach by the *x*-rays, by administering food mixed with bismuth, and by observation with a fluoroscope. He found that the contractions start in the middle of the stomach and pass toward the pylorus at regular intervals. The pyloric end of the stomach lengthens out and the peristaltic waves increase during advanced digestion. At intervals the pylorus relaxes and the contraction squeezes part of the chyme into the duodenum.

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[1] American Journal of Physiology, 1898.

Cannon[1] finds that carbohydrates pass out soon after ingestion and require only about one-half the time that proteids do for gastric digestion. Fat when taken alone remains for a considerable time in the stomach, and if combined with other food its exit is delayed. It is believed that chemic stimuli, such as hydrochloric acid, control the opening and closing of the pylorus.

If atony of the stomach be present, motor insufficiency and stasis result, and the latter favors fermentation and putrefaction. It has been demonstrated that the presence of hydrochloric acid in considerable quantities does not prevent these conditions if stasis be present.

INTESTINAL DIGESTION

Under normal conditions, when the chyme enters the duodenum its reaction is acid. It is here subjected to the influence of the bile, pancreatic juice, and intestinal secretions, all of which, in their action, have a more or less close interdependence upon each other.

Bile.—The bile in the intestine precipitates the pepsin from the chyme. This, however, is soon dissolved. Others hold that albuminoids are precipitated from the chyme, together with the pepsin, and are then more readily absorbed. It does not have a deleterious effect on the pancreatic digestion.

The bile is one of the chief factors in gradually *altering the reaction of the chyme to neutral or alkaline*, and *it strongly supplements the action of the pancreatic juice in emulsifying fats, and its absence or diminution lessens this function.*

Pawlow believes that it may augment slightly the effects of the other pancreatic ferments.

One of its chief functions *is undoubtedly excretory*, and through it many of the useless products of metabolism are eliminated. When absorbed into the system it acts as a poison and produces a definite toxemia.

The bile is a clear, tenacious mucoid fluid with an alkaline reaction, consisting of water, bile-acid salts (glycocholic and taurocholic acid), pigments (bilirubin and biliverdin), mucin, cholesterin, lecithin, soaps, fats, etc.; about 500 to 600 cc. are excreted in twenty-four hours.

Wm. H. Porter[2] believes that the precipitation of the pepsin from the chyme *through the action of the bile* is necessary for the action of the pancreatic ferments, and the enterokinase of the intestinal juice, as the pepsin, as long as it remains active, inhibits their action.

Some consider that it has antifermentative and antiputrefactive powers, and that it helps to maintain the nutrition of the epithelial cells. Lindenberger has shown experimentally that a small amount of bile combined with .05 per cent. of lactic acid prevents putrefaction in an infusion of pancreas, while lactic acid alone has no effect.

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[1] American Journal of Physiology, 1904.

[2] Indicanuria a Danger Signal, Postgraduate, 1907.

Von Noorden holds that bile has no antiseptic action.

Clinically, we at times see cases of indicanuria with the presence of bile in the urine, with light-colored stools in which there is no *permanent disappearance of indican*, though there is a temporary improvement after the use of calomel, etc., until the biliary excretion into the intestine is restored to normal. The internal use of the bile-salts, which can be given as inspissated bile, seems to be of some value. Bile possesses slightly purgative qualities.

The liver has special functions. Many of the substances taken up by the digestive process are stored there until used in the system, namely, some of the peptones and sugar in the form of glycogen. It excludes some poisonous matters from the circulation. Thus it is the chief organ for the removal of indol and poison, such as curare or of various autotoxins. Urea is also formed in the liver.

Pancreatic Juice.—The pancreatic juice is the principal factor of digestion in the intestinal canal. It is clear, colorless, alkaline, sticky and odorless, quite albuminous, containing water, solids, proteids, and inorganic matter.

The most important constituents are the three ferments:

Amylopsin, an amylolytic enzyme, which converts starch into maltose, and still further into glucose. Cane-sugar is converted into grape-sugar, while milk-sugar is unchanged. Some of the cellulose ferments form marsh-gas and various acids. The activity of pancreatic diastase is increased by very small quantities of acids (Chittenden).

Steapsin, a lipolytic ferment. This acts upon fats by splitting them into fatty acids and glycerin; and this action is increased by the bile. The fatty acids combine with the alkalis in the intestine to form soaps, which aid in the emulsifying of fats, and thus promote their absorption. The emulsification occurs in an alkaline medium, or in contact with the alkaline secretion of the mucous membrane.

Trypsin, a proteolytic ferment, changes the proteids into albumoses and peptones. There is probably a rennet-zymogen also.

The ferment acts in a neutral or alkaline medium, though slight degrees of acidity seem to favor it. Indol is a product of intestinal putrefaction, and not from the action of the pancreatic ferment, as was formerly supposed. The same is true of hypoxanthin.

The secretion of the pancreatic juice is not reflex, but is due to direct excitation of the cells of the pancreas by secretin (Starling). This is formed in the mucous membrane of the *duodenum* and *jejunum* and reaches the pancreas by the blood-stream. The passage of the chyme over these portions of the small intestine stimulates the production of secretin or, rather, splits it off from "presecretin," which is present in the mucous membrane.

The **intestinal juice** (**succus entericus**) consists of water albumin, mucin, and salts. Ptyalin and an inverting enzyme hav been discovered therein. It seems to neutralize the acids forme by the fermentation of the carbohydrates and the presence of muci shows it to be of service in aiding peristalsis.

Pawlow's experiments demonstrate that it augments the activit of the pancreatic ferments, especially of trypsin. The fat-splittin and amylolytic pancreatic ferments are augmented by the succu entericus from all parts of the small intestine, while that from th duodenum augments chiefly the proteolytic ferment.

Pawlow found enterokinase, a ferment of other ferments, which believed to be necessary to excite intestinal ferments into activit

Erepsin, another ferment, is also present, which transform hemi-albuminose into other bodies.

Organized Ferments.—There exist in the intestine fermentati and putrefactive changes produced by micro-organisms.[1] At birt the gastro-intestinal tract is sterile, but rapidly, by the ingestion food and through the air and by the anus, bacteria of various typ enter this tract.

Herter[2] has estimated their number at one hundred and twent six billions for the daily human excreta. Many of them are no long living. He considers the chief function of the obligate bacter (Bacillus lactis aërogenes, Bacillus coli, and Bacillus bifidus) to their capacity for checking the development of other types of orga isms capable of doing injury, though they themselves under certa conditions may produce much harm. Many other varieties a described. Some observers believe that there is a so-called norm fermentative process which aids in the digestion of cellulose, thoug Bergman claims there are enzymes (intracellular) which decompo it.

The fermentative processes in the small intestine caused by t action of bacteria on the carbohydrates (Bacillus lactis aërogene leads to the formation of ethyl alcohol and various organic acid such as lactic, acetic, paralactic, succinic, biliary acids and albumi peptone, mucin, sugar, etc. These organic acids are believed prevent putrefaction within the intestine, to partly check the decom position of the carbohydrates, and to aid in producing intestin peristalsis, which render putrefaction less likely. The lactic ac in koumyss, matzoon, bacillac and lactone milk, and even in pla milk (to a slighter degree), is believed to lessen putrefaction. the lower jejunum and ileum the reaction is acid.

When the intestinal contents pass into the colon the reacti becomes alkaline, fermentation stops, putrefaction begins, and t fecal odor appears. The colon bacilli are marked factors in this process. The decomposition of the albuminates caused by bacteria goes much further than that produced by pancreatic digestion. Albumoses, peptones, lysin, ammonia, amido-acids, etc., are produced in both cases, but with putrefaction the process goes further and we have new products formed, such as indol, skatol, paracresol, phenol, various acids and gases, such as sulphuretted hydrogen, marsh-gas, carbon dioxid, etc.

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[1] Proceedings American Medico-Psych. Association, April 21, 1905. So Observations on the Relations of the Gastro-intestinal Tract to Nervous a Mental Diseases (Kemp).

[2] Bacterial Infections of the Digestive Tract.

Some of these products of decomposition are eliminated unchanged in the urine, such as the oxyacids, others, like the phenols, after further oxidation, and others, like indol and skatol, after combination with ethereal sulphuric acids. For example, indol forms an indoxyl-potassium sulphate or indican, and is so eliminated in the urine; and thus may be an indication of the amount of putrefaction occurring in the intestine.

The causes of indicanuria are various, such as excessive proteid diet, catarrh of the small intestine causing alterations in the mucosa and increased intestinal putrefaction therefrom, typhoid, cholera, a pus-cavity, constipation, alimentary putrefaction, decrease of normal digestive fluids, intestinal obstruction, and peritonitis. Certain drugs, such as salol, salophen, and creosote, will give nearly similar reaction, while urotropin will cause its disappearance. These possible conditions must all be considered.

As the intestinal contents pass through the large intestine they become thickened through the absorption of fluids and are at last eliminated as feces. These comprise the remains of undigested material, excretory material from the intestines, and many micro-organisms.

The quantity of feces depends upon the character of the food, being greater after a vegetable diet. The average amount after a mixed diet is about 100 to 150 gm.

The reaction of the feces is ordinarily alkaline, though occasionally variable. The odor is chiefly due to skatol, and the color is a light or dark brown.

Absorption from the Intestines.—Absorption chiefly occurs in the small intestine.

The *proteids* are usually changed into albumoses and peptones before absorption occurs. Albumin may be absorbed as such, though not as quickly.

Absorption of the albumoses and peptones takes place by the capillaries and not by the lacteals. They are reconverted into albumin before reaching the blood-current, probably either by the epithelial cells or leukocytes. It has been shown experimentally that they do not reach the blood as peptones.

Albuminates from animal food are more completely absorbed than those from vegetable food, on account of the indigestibility of the cellulose and the increased peristalsis caused by the latter.

Absorption of the Carbohydrates.—These are chiefly absorbe as monosaccharids through the capillaries of the villi, enter the live through the portal vein, and are retained as glycogen for use in th animal economy.

If sugar is absorbed in excess, it may enter the general circulatio and be excreted by the kidneys, so-called alimentary glycosuri It may also cause diarrhea.

Carbohydrates, as starch, are absorbed without difficulty.

Glucose, levulose, and galactose are absorbed as such, whi cane-sugar and maltose are first changed to these products. Mill sugar is unchanged and absorbed as such, or undergoes lactic-ac fermentation.

Absorption of Fats.—The greatest amount is absorbed as emulsion (both fats and fatty acids), though some absorption tak place in the form of soaps. They become neutral fats after absor tion. They enter the lacteals probably through the action the epithelial cells of the intestinal wall and reach the thorac duct.

The absorptive power for fat in the small intestine is considerabl probably over 300 gm. per day. Olive oil and butter (fats with low melting-point) are absorbed more quickly than mutton fat, f instance (fat with a high melting-point), and a free fat, such as butt is taken up more quickly than bacon, which contains considera connective tissue.

Water, salts, some of the secretory juices, and bile are readi absorbed.

Disease or removal of the pancreas stops the absorption of fa except of milk, of which part is absorbed in emulsified form.

Absorption in the Large Intestine.—Water, fluids, and sa are well absorbed, in fact, markedly absorbed, as is noted by t change in the character of the intestinal contents. Albumin a carbohydrates are absorbed in considerable amount and fats in sm quantities. Advantage is taken of this fact for the employment nutritive enemata.

Intestinal Peristalsis (**Motor Function**).—The contents thoroughly mixed by the movement of the intestines, and t residuum left after digestion is expelled through the anus.

There are three types described:

The intestine contracts at a certain point and then relaxes, a continues this in successive segments progressively toward the an pushing the contents forward (*ordinary peristaltic movements*).

Oscillating movements, by which the coil is moved to and along the mesentery, with no particular contraction. The conte are mixed up by these movements and not propelled forward.

Rotary movements, by which a coil contracts in a circular direct rapidly along the intestine for 15 or 20 cm. in a violent manner.

The last is usually pathologic and occurs when there is c siderable gas, after indiscretions in diet, or in stenosis. It is observed only in the small intestine.

Peristaltic action is much more rapid in the small intestine. After ingestion of a small meal the stomach becomes empty in about two hours, the small intestine in the same length of time; but in the large intestine it takes at least twenty hours before the contents are expelled.

Nothnagel has never seen a physiologic antiperistalsis (reversed peristalsis) beginning from the anal direction upward toward the stomach, though some[1] describe an intermittent antiperistalsis occurring in the ascending colon. It is said that strong injections of salt water into the colon will produce this effect.

Nervous Control of Peristalsis.—Auerbach's and Meissner's plexuses are probably the automatic centers for peristalsis, but there are central agencies. For example, fright or excitement may cause diarrhea.

The splanchnic nerve contains inhibitory fibers for the control of intestinal peristalsis.

Ehrmann claims that the longitudinal muscles are stimulated by the splanchnics and inhibited by the vagus, and the circular muscles stimulated by the vagus and inhibited by the splanchnics.

The chyme acts as the normal stimulus to peristalsis through the nerves. Toxic material that has been ingested or developed in the intestinal canal, indigestible food, organic acids from excessive fermentation, and too hot or too cold drinks, may overstimulate the peristaltic action and be the cause of diarrhea.

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[1] W. B. Cannon, Amer. Jour. Physiol., vol. vi., p. 253, has demonstrated by the Röntgen rays that antiperistaltic movements occur normally in the cecum, ascending and transverse colon, thoroughly churning and mixing the food, and bringing it in more perfect contact with the absorbing surface of the colon.

CHAPTER III

INTERROGATION OF THE PATIENT (HISTORY)

In every case suffering from symptoms pointing to the gastro-intestinal tract, before the physical examination is undertaken, the patient should be carefully interrogated as to his general history and the past and present symptoms.

I shall briefly indicate the form of taking and preserving the history of such cases:

Date.................. Diagnosis............... No. of patient............

Name..............................
Age...............................
Sex...............................

Nativity.............................
Occupation...........................

Family History

Tuberculosis:
Syphilis:
Diphtheria:
Scarlatina:
Typhoid:
Gout:

Rheumatism:
Malaria:
Influenza:
Nephritis:
Heart disease:
Disease of liver:

Habits

Tea—cups:
Coffee—cups:
Tobacco:
Alcohol:

Sexual excess:
Mastication:
Character of food:

Past History of Present Complaint

Began:
Duration:

Onset—
- Sudden:
- Gradual:

Probable cause:

Symptoms—
- Progressed:
- Same in character:
- Changed in character:

Loss of flesh—
- Present:
- Increasing:
- Absent:

Bowels—
- Constipation:
- Diarrhea:
- Alternating:
- Regular:

- Mucus:
- Blood:
- Odor:

Present Condition and History

Headache—
Character:
Location:
Time of:

Vertigo—
Nervousness:
Drowsiness:
Sleeplessness:

Appetite—
Good:
Anorexia (loss):
Time:
Parorexia (perversion):
Bulimia (canine hunger):
Polyphagia:
Akoria:

Thirst:

Taste—
Normal:
Bitter:
Time:
Sour:
Sticky:

Deglutition:

Dysphagia—
With solids:
With liquids:

Abnormal sensations—
Bloating:
Fulness:
Time:
Pressure:
Weight:

Belching—
Quantity:
Time of:
On full stomach:
On empty stomach:
Odor:

Regurgitation—
Water-brash:
Sour:
Time:

Rumination:

Pyrosis (heart-burn)—
Time:
Duration:

Pains—
Location:
Cardialgia:
Gastralgia:
Character:
Time of appearance:
Duration:
Affected by position:
Affected by food or drink:
Circumscribed:
Diffuse:
Radiating:
Sudden:
Slow:
Relieved by pressure:
Increased by pressure:

Local tenderness—
Position:

Nausea—
Time:
Affected by food:

Vomiting—

Time:
Frequency:
Quantity:
Character of:
Odor:
Taste:

Blood and its character:
Bile:
Mucus:
Easy:
Difficult:
Relief of pain by:

Stools—

Regular:
Constipation:
Diarrhea:
Alternating constipation and diarrhea:
Number of movements:

Time of appearance:
Undigested food:
Mucus:
Blood:
General character:
Bile:

General health and strength—

Loss of weight:

Symptoms Referable to Circulatory System

Symptoms Referable to Nervous System

Chief Complaint

After the physical examination has been made, the re should be incorporated with the history, as should also the secured from examination of the gastric contents and stool.

General Physical Examination Comprises—

Tongue: *Uvula:* *Tonsils:* *Pharynx:*
Teeth:
Eyes—as to difficulty in reading or headache therefrom; exophthalmos.
Nose—as to nasal discharge.
Ears—as to deafness.
Heart:
Lungs:
Liver:

Stomach—

Position:
  Normal:
  Dilated:
  Gastroptosis:

Tenderness:
Motor function:
Typanitis:
Gastric analysis:
Tumor:

Spleen:

Kidneys—

Position:
Urine:

Intestines—

Position:
Tender points:
Thickening:

Tympanitis:
Borborygmi:

Rectum --
Local examination:
In some cases microscopy and fermentation test of stool.

Nervous system:

Weight of patient:

Examination of genital organs—in some cases.

CHAPTER IV

GENERAL METHODS OF PHYSICAL EXAMINATION

As patients who complain of digestive disturbances may suffer from disease of other organs which may be the cause of the symptoms, a thorough physical examination should be made in every case.

The first step should be an examination of the heart and lungs. The character and rapidity of the pulse and respiration should be noted and a specimen of urine requested for analysis.

In all *acutely* commencing processes pointing to *the digestive tract, the temperature should be taken.*

GENERAL INSPECTION

The general appearance of the patient may afford valuable information. With cancer, there is often the sallow and emaciated appearance (cachexia) with anemia; with gastric neurosis, the patient may often appear rosy and well nourished; while with ulcer there is frequently marked anemia and the face may have the appearance of suffering. Protrusion of the eyeballs, with inability of complete closure of the lids, taken in connection with tachycardia, are suggestive of Graves' disease, even though no thyroid enlargement be present. The skin should be inspected for eruptions and the exanthemata.

Oral Cavity.—This should be carefully inspected. Defective and carious teeth or inflammation or abscess of the gums may give rise to gastric disorders. Disease of the posterior nares or middle ear, with resulting discharges passing into the pharynx, may be factors.

The tongue was formerly regarded as a mirror of the stomach, but it can hardly be so considered, as there are some gastric affections in which the appearance of the tongue is normal; while in smokers, for example, the tongue may be coated and yet no gastro-intestinal disturbance be present. A thick gray or grayish-yellow, moist, coated tongue showing indentations is suggestive of chronic gastritis; while with ulcer it may be dry and red with a white median stripe, or smooth and moist, or slightly furred.

The condition of the mouth, smoking, drinking, and the teeth, have a decided bearing on its appearance. This is true in reference to the odor of the breath. I hardly deem the tongue diagnostic in diseases of the gastro-intestinal tract except in association with other symptoms.

With the typhoid state, we have the narrow tongue, with the deep median fissure, thickly furred, the tip and edges being red and denuded, or the dry brown, fissured, and tremulous tongue; while with scarlet fever and in some other acute specific infections there is the so-called strawberry or raspberry tongue, with bright red projecting papillæ.

The uvula is sometimes elongated, and may cause reflex digestive disturbances. In the pharynx and tonsils there may be inflammatory conditions of an acute or chronic type which influence the case.

Inspection of the neck is important. Enlarged thyroid with tachycardia is diagnostic of Graves' disease, and a swelling to the left of the larynx, which increases in size after the ingestion of food, is suggestive of a diverticulum of the esophagus.

Examination of the Esophagus.—The cardinal symptom of esophageal disease is dysphagia, with or without regurgitation. The object of examination is to determine whether a stricture or a diverticulum is present.

Palpation of the esophagus is possible in the neck, usually on the left side behind the trachea. A tumor found here may be a diverticulum distended with food or fluid. It is sometimes possible to empty it by the exertion of pressure.

A brawny swelling, in some cases with subcutaneous emphysema may result from perforation or rupture of the esophagus, with inflammation, which proceeds to suppuration. An abscess in this locality may be due to caries of the vertebræ.

Auscultation of the esophagus is often of service. Place the stethoscope posteriorly, to the left of the spine, at the level of the sixth dorsal vertebra, and at a signal let the patient swallow a mouthful of water. At the instant of swallowing the deglutitory sound is heard, followed in six or seven seconds by the esophagea bruit, which resembles the sound one hears when swallowing saliva Three to five seconds later there is a second sound caused by the fluid entering the stomach or by regurgitation of air. If the first sound is delayed longer than seven seconds, or replaced by a splashing or gurgling noise, or if the second sound is delayed longer than five to twelve seconds, partial stenosis may be suspected. If both sounds are absent, there is probably nearly complete or complete stenosis

Gurgling sounds lasting several minutes and heard along the left side of the spine are probably due to contractions in a diverticulum or in the dilated portion of the canal above a stricture.

Instrumental examination of the esophagus is made by flexible stomach-tubes of various sizes, the safest method, or by flexible solid bougies or sounds.

The esophagoscope is of chief value in locating foreign bodies and affording direct means of removal.

In the use of the sound one must remember that it is 6 inches from the incisors to the commencement of the esophagus at the cricoid cartilage; 9 inches to the crossing of the left bronchus, and 16 inches to the cardiac orifice of the stomach. There is normally some constriction of the esophagus at these three points.

Obstruction to the passage of the tube may be due to esophageal spasm, but by waiting this will usually subside. I have found that in some of these cases a large tube can be passed more readily than a small one. If the tube pass readily on one occasion and refuses to pass on another, it has probably slipped into a diverticulum, or the latter has filled up and by pressure prevented the passage of the instrument.

If the obstruction is permanent, one must decide whether it is due to stricture (narrowing) or external pressure, such as from aneurism, tumor, or enlarged glands. Syphilis, cancer, and contraction following burns from acids or alkalis are the principal causes of stricture. Congenital stricture is rare.

If stricture be present, the locality, caliber, and permeability must be determined.

Locality.—Pass the tube to the strictured point, nip it close to the incisor teeth, and measure the distance from here to the entering tip of the tube after withdrawal.

Caliber.—Sounds of varying diameters will determine the caliber by finding one which will pass the obstruction. By the esophageal bruit and the use of a somewhat rigid sound one can tell whether or not the obstruction is *permeable.*

Occasionally small portions of new growth may be found in the openings of a fenestrated tube. Blood shows ulceration or erosion.

Contra-indications.—The tube should never be passed if there is aneurism of the thoracic aorta or recent vomiting of blood.

Examination of the Abdomen.—The special methods of physical examination of the stomach and intestines are described in the parts of this volume devoted to these subjects. It seems advisable to refer to the general methods of examination of the abdomen and the other viscera.

Anatomic Landmarks.—The ensiform appendix and down-curved arches of the ribs constitute the upper bony landmarks. The iliac crests, anterior superior spines of the ilia, and the symphysis pubis in the median line are the lateral and lower boundaries.

The linea alba lies between the recti muscles in the median line, runs from the ensiform appendix to the pubic symphysis, and is visible as a groove above the umbilicus.

The umbilicus is somewhat variable in its position, though usually lying about 2 inches above the bispinal line drawn transversely between the anterior superior spines of the ilia. The recti muscles lie on each side of the linea alba and are bounded externally by the lineæ semilunares, which run with an outward curve from the lowest part of the seventh rib to the pubic spines. These lines lie on each side about 3 inches from the umbilicus.

Topographic Areas.—In order to describe the situation of organs or lesions, the surface of the abdomen is divided into regions, of which the method depicted in Fig. 15 is in general use.

There are nine regions divided off by four lines, two horizontal and two vertical. The horizontal lines are the infracostal or subcostal, drawn transversely at the level of the inferior borders of the tenth ribs, and the bispinal line, connecting the anterior superior spines of the ilia. The vertical lines pass through the center of Poupart's ligament on each side and are downward prolongations of the mammillary lines of the thorax. The boundary lines between the epigastric and hypochondriac regions correspond with the costal margins, and the iliac regions correspond to the so-called inguinal regions.

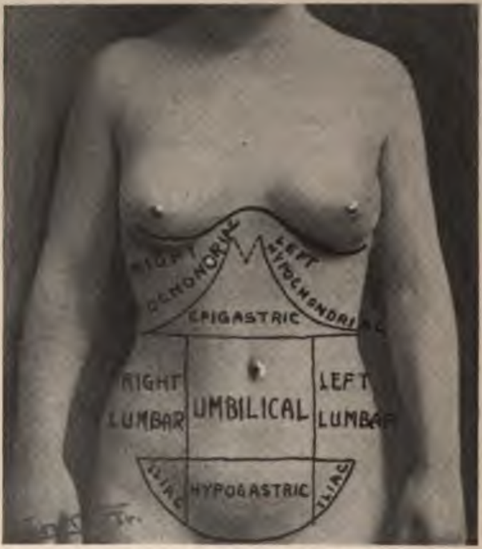

Fig. 15.—Topographic areas of the abdomen.

A second method of dividing the abdomen is by a vertical and transverse line through the umbilicus, forming four quadrants.

In reference to the various landmarks, one often measures distances by the finger-breadth. The average finger-breadth is ¾ inch; two, 1¼ inches; etc.

A hand-breadth averages 3½ to 4 inches.

Preparation of the Patient and Technic.—The patient should preferably lie in bed in the dorsal position, quite flat, with the head on a single thin pillow. The same posture should be assumed if the examination be made on the office table. The bedclothing should be drawn well down, except the sheet, under cover of which the nightdress or undershirt should be drawn up to the lower sternum, and the sheet then folded down to a short distance above the pubes.

The sheet may not be required in the office, though generally preferable.

The patient should lie as symmetrically as possible, with the pubic spines at the same level and a good light secured. It is of value to make an observation also in the *sitting* or *standing position*, as *prolapse of the abdominal walls* or *viscera* can thus be more clearly seen.

Inspection.—Inspection should be from the front, sides, and back. It is of great importance. With excessive abdominal distention the skin is smooth, shining, and stretched. Copper-colored, scaly, somewhat circular spots are significant of secondary syphilis; and whitish streaks, or striæ, of long-continued distention, such as from pregnancy or ascites.

Typhoid eruption or exanthemata may in some cases be in evidence. Glandular enlargements in the groin or old scars are suggestive of venereal infection. Inguinal or femoral hernia may be observed.

Inspection of the blood-vessels often affords valuable information. Enlarged veins radiating from the umbilicus (the caput medusæ) are significant of portal obstruction, cirrhosis, or tumor of the liver. General enlargement of the abdominal veins may be present in similar conditions, or from pressure on the venæ cavæ by thoracic or abdominal tumors. If a dilated lateral vein is present running up the right midaxillary line, it should be emptied by massage, and the *method of its refilling* carefully observed. If the portal vein, or the inferior vena cava, is obstructed, the direction is upward; but if the superior vena cava is pressed upon, the direction is downward.

Distention of the veins in the pubic region alone shows some probable obstruction below the liver.

Enlarged epigastric arteries are diagnostic of obstruction of the aorta or iliacs.

An umbilicus that protrudes is suggestive of hernia, ascites, pregnancy, or some form of abdominal distention.

Absence of respiratory abdominal movements, with accentuation of thoracic respiration, is significant of peritonitis.

Peristaltic unrest (visible peristalsis) is usually diagnostic of stenosis of the pylorus or intestines or of intestinal obstruction. It may rarely occur normally in thin persons.

One can at times infer the site of the obstruction by the location and character of the peristalsis. The waves run in the stomach from left to right and in the transverse colon from right to left. If the obstruction is near the ileocecal valve, the *swollen* and *moving coils* of the intestine lie one above the other in the central part of the abdomen (ladder pattern).

If the constriction is lower down in the large intestine, the distention is chiefly visible in the course of the colon (in the circumference of the abdomen). A recurring protuberance at one point, disappearing with a loud sound, is probably near the point of stenosis.

Protrusion from tumor can at times be observed.

Method of Abdominal Palpation.—The right hand should be warmed and laid flat upon the surface of the abdomen, the physician sitting to the right of the patient (Fig. 16).

Palpation should be at first by somewhat circular pressing movements, sliding the skin over the parts beneath, and passing from one portion of the abdomen to another. One should not poke suddenly with the finger-tips. Gradually deeper localized palpation may be made with the finger-pulps to determine the presence of tender spots, or the shape, size, and mobility of existing masses or swellings.

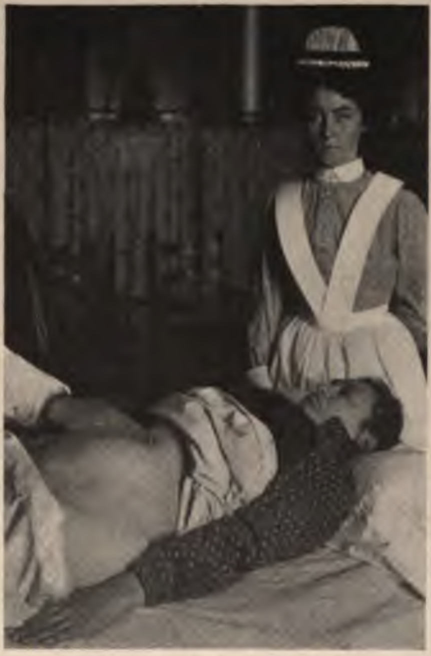

Fig. 16.—Abdominal palpation.

The facial appearance of the patient rather than the verbal expression tells whether true pain is present. Tenderness suggests inflammation or ulceration. McBurney's point should always be explored for appendicitis.

If firm pressure elicits tenderness, it is apt to be real and deep seated, rather than a surface lesion or hyperesthesia.

If hysteria is suspected, the patient's attention can be diverted by pressing on a different part of the surface with one hand, while the other hand explores the original seat of pain complained of. Absence of true tenderness is thus frequently revealed.

If the abdominal muscles are contracted, the knees and thighs should be flexed and a pillow placed beneath the head and shoulders to secure relaxation. The *flexion of the lower limbs* I believe *preferable in every case.* Deep and rapid respirations at the end of expiration relax the muscles momentarily and render examination more easy. This method aids in differentiation, whether the mass felt is due to contraction of the belly, of a muscle, such as of the rectus, and whether a tumor is movable with respiration. Howard Kelly recommends vibratory movements with the fingers while palpating.

In some cases reinforced palpation, the left hand exerting pressure over the right, as in Fig. 17, is of value, especially in examination of the deeper organs. During examination forced respiration should be taken, and at each expiration the abdominal wall should be pressed upon firmly, maintaining during inspiration the ground which has been gained. This method is of special service in determining a chronic enlarged appendix, as suggested by Edebohls and R. T. Morris.

If there is fluid in the peritoneal cavity and one desires to palpate an organ which is obscured by its presence, sudden deep pressure with the finger-tips ("dipping") will displace the fluid. In some cases a general anesthetic may be necessary for a thorough examination.

All the regions of the abdomen should be explored, the *umbilical*, the *inguinal*, and *femoral regions* being examined for hernia.

When the lateral regions are examined, both hands should be employed, one being slipped under the body so as to make forward pressure between the last rib and iliac crest, thus pushing forward the structure against the examining hand in front.

At times it may be of service to examine in the knee-elbow position, or with the patient standing and leaning forward, supporting himself with the hands by a table or chair. If he is very fat, it is often useful to have him turn partly on the side, thus "spilling" the intestines and fatty abdominal walls away from the region under investigation.

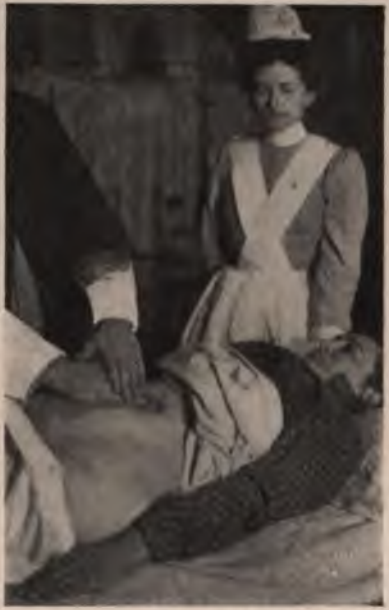

Fig. 17.—Reinforced palpation.

Digital rectal and vaginal examination are very necessary in many cases, especially when the lesion is situated in the lower third of the abdomen. Rigidity of one or both of the recti muscles is of great diagnostic importance, being significant of peritoneal irritation, local peritonitis (if one muscle be involved), or general peritonitis if both recti and all the abdominal muscles are affected.

Occasionally a rigid rectus is found on the side of a pneumonia or diaphragmatic pleurisy.

The upper segments of one or both recti may be rigid in abscess of the liver, or in subphrenic abscess, or of the right rectus in acute cholecystitis; the right rectus, especially the lower segment, in appendicitis; and the left rectus in diverticulitis, or in left-sided pelvic inflammation.

Mensuration of the circumference of the abdomen at the level of the umbilicus and of the length of its anterior wall from the ensiform to the symphysis, are of use in noting the increase of ascites or tl growth of a large tumor. An uneven protuberant surface is cha acteristic of a malignant growth; an even surface is more often four in benignant neoplasms or intussusception. A fecal tumor ca usually be indented and as the finger is raised the intestinal wa slips from the mass.

Percussion of the Abdomen.—With the exception of pulmona resonance, which we note in defining the upper limits of the stoma and liver, and splenic and hepatic dulness, the normal abdomen tympanitic. From the presence of food in the stomach or fec accumulation in the intestines there are variations, with resulti dulness or even flatness. The percussion note over the sigmo flexure and lower part of the descending colon is quite frequent dull, owing to the tendency to fecal accumulation in these regior

In general, we may say that the pitch of the resonant note vari with the size of the air space and the degree of tension of the co taining cavity; the smaller the air space and the greater the tensic the higher is the pitch. Hence, the empty stomach and colon wou afford a lower pitched note than the small intestine.

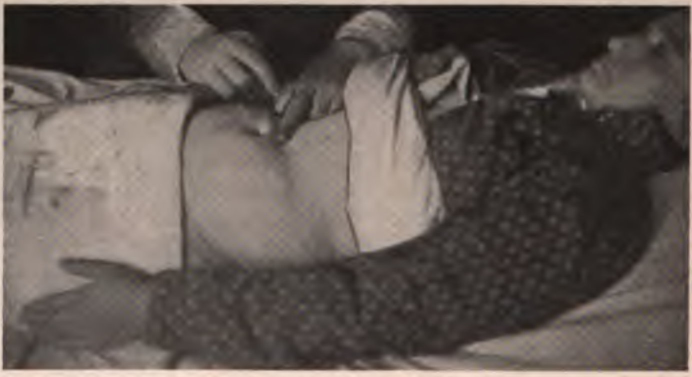

Fig. 18.—Simple percussion.

The presence of food and liquid in the stomach modifies t results of percussion, as do also feces in the large intestine. F example, with an empty stomach we have tympanites; and th quite frequently a change in note over the transverse colon to duln or even flatness; or with the partially full stomach and empty int tine, tympanites above, then dulness or flatness over the conte and intestinal tympanites below. It is well, therefore, to have t large intestine cleared out by enema before examination. Practica we find in many cases stomach tympanites with change in note ov the colon due to some contents.

Among the best methods of percussion are simple percussi with the finger or hammer, flicking percussion, auscultatory p cussion, and the "scratch method" of auscultation.

In simple percussion, the middle finger of the left hand should be laid flat on the abdomen (the pleximeter) and the mid-finger of the right hand, bent at right angles, should be employed as the plexor, as depicted in Fig. 18. The other fingers and thumb should be folded into the palm of the hand.

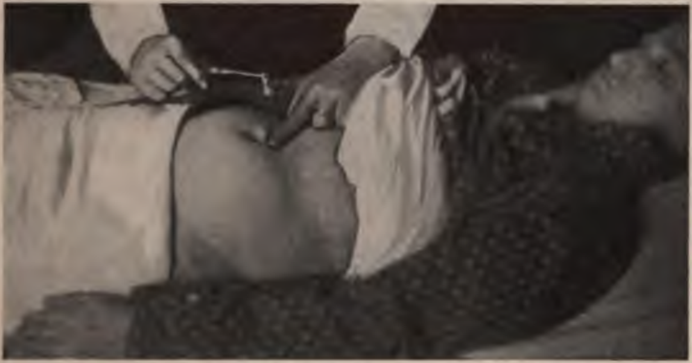

Fig. 19.—Percussion hammer.

In Fig. 19 is shown the method with the percussion hammer and the correct position. The finger is preferable as a pleximeter, as the rubber instruments interfere with the sounds.

"Flicking percussion" is useful in detecting slight degrees of dulness.

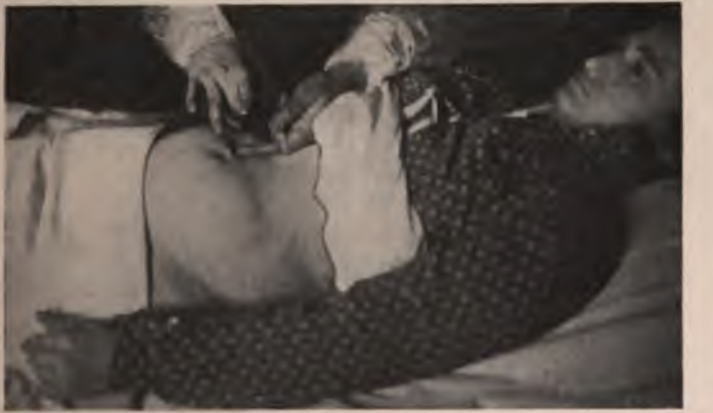

Fig. 20.—Flicking method of percussion.

The forefinger or middle finger of the left hand should be placed nail downward on the surface; the middle finger of the right hand is well flexed, so that the nail is pressed against the palmar surface of the thumb. It is then suddenly allowed to escape, so as to strike sharply against the palmar surface of the finger lying on the abdomen (Fig. 20).

Auscultatory percussion is probably of greatest value in *outlinin contiguous air-containing viscera.*

The stethoscope is placed over the organ and the normal not secured by percussion close to the instrument. Then percussion i carried out, beginning at some distance, from above, below, an laterally, and the change of note observed.

This method and the "scratch" method will be described lat in outlining the position of the stomach and intestines.

If the percussion note of a deep-seated mass is to be elicited, t pleximeter finger must be pressed slowly and firmly down in ord to push aside or compress air-coils of intestine which would mas the note.

If dulness is present where it should not exist, it should ascertained whether it disappears or shifts with changes in t position of the patient, *i. e.*, whether it is fluid.

If the distention is due to ascites (fluid), the center of the abdom is flattened and the lateral and dependent portions bulge outwar providing the fluid is not excessive. If it is very great, the abdom is arched and prominent, the umbilicus is bulging or stretched, a the shape is not changed when the posture is altered.

On percussion, the flanks are dull and the center of the abdom tympanitic, as the intestines float to the highest point. Unless t quantity of fluid is excessive the line of dulness changes its positio as the patient is turned on the side the fluid gravitating to the low point and being replaced by the tympanitic intestine. The upp flank, previously dull, is now tympanitic. If a small amount of flu is suspected, percussion in the umbilical region in the knee-elb position will give dulness, when it was tympanitic in the dor position.

Fluctuation may be elicited if there is considerable fluid.

The ulnar edge of a nurse's or assistant's hand should be press firmly on the linea alba, to cut off muscular vibrations.

One hand of the examiner is placed upon one lateral abdomi wall, while he should tap sharply with the fingers on the opposite si If fluid is present, a transmitted wave—at times visible—will felt by the palpating hand.

With tumors, enlargement of the abdomen is not symmet Percussion does not show the uniform resonance of gas nor lateral dulness and central tympanites of fluid, and palpation de onstrates the solidity of the mass.

With gas, the abdomen is arched and tense, universally ty panitic, and fluctuation cannot be obtained.

Sources of Error.—The segments of the recti muscles wh contracted may simulate a small tumor.

By insinuating the tips of the fingers under the edge of apparent tumor and having the patient raise the head and should the muscle is felt to contract and thicken.

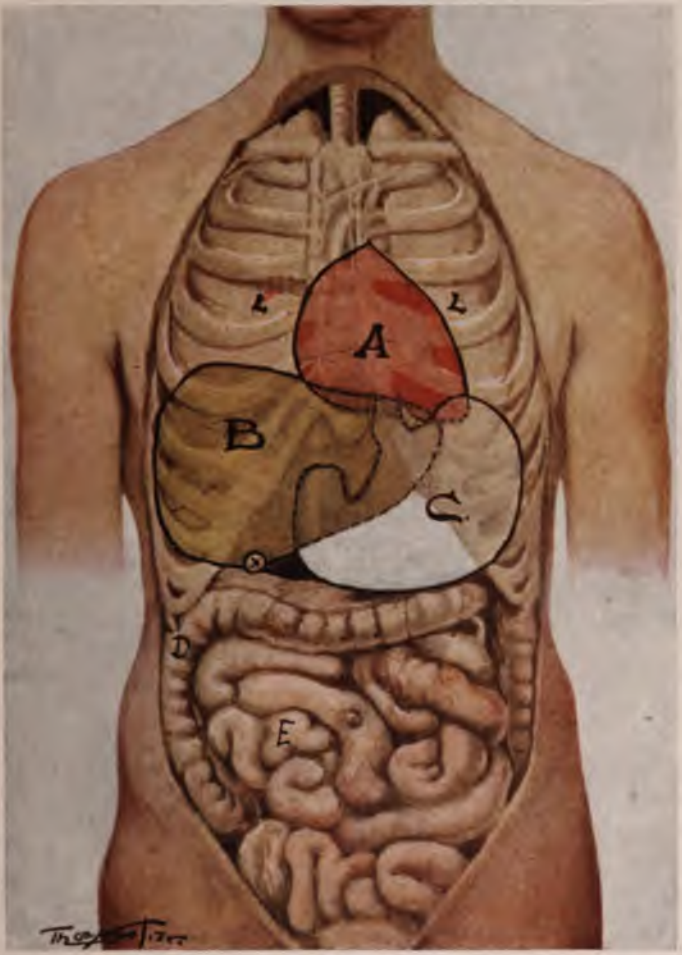

Fig. 21.—Relations of the viscera. Anterior view: *L*, *L*, Lungs; *A*, heart; *B*, liver; *C*, stomach; *x*, gall-bladder; *D*, colon; *E*, small intestine.

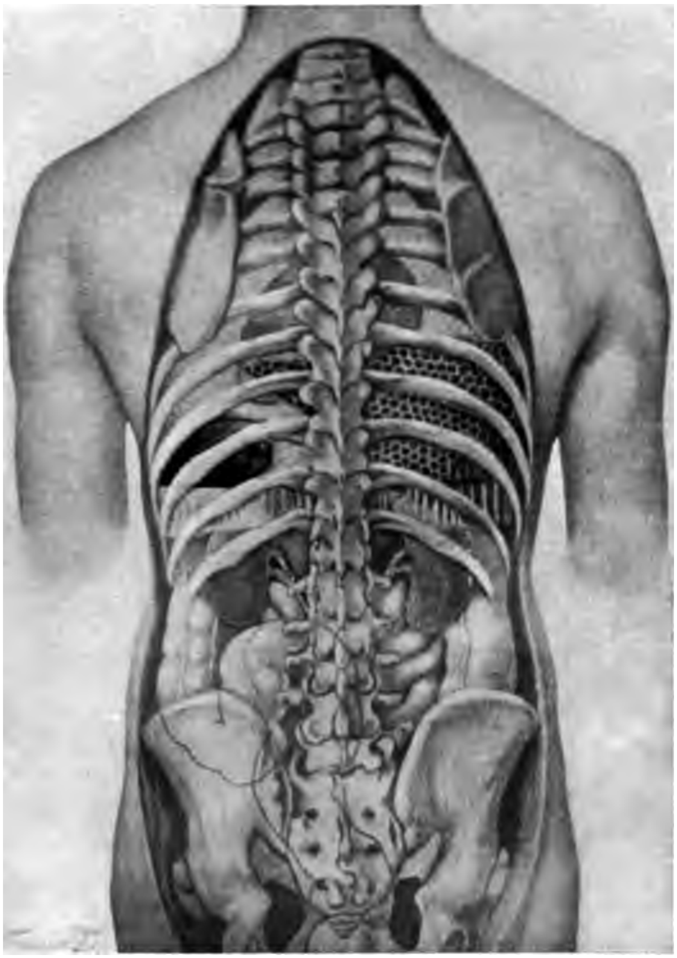

Fig. 22.—Relations of the viscera. Posterior view.

A localized contraction of the abdominal muscles or a persistent gaseous distention of a portion of the intestines ("phantom" tumor) may be deceptive. These occur, as a rule, in hysteric women and are dull or tympanitic, depending on the above conditions. They disappear during rapid forced respiration or under anesthesia.

Fitz believes that some "phantom" tumors are congenital or acquired dilatation of the colon.

Auscultation of the Abdomen.—*Sounds in the Abdominal Cavity.*—In the healthy intestines there are always bubbling or gurgling sounds heard on auscultation. The entire absence of sound is significant of intestinal paresis.

With mechanic obstruction the sounds are usually increased in intensity and number.

In intestinal paresis, usually due to peritonitis, the heart and respiratory sounds may be audible over the entire abdomen. This is not true in tympanites due to other causes. Crepitation or friction sounds are at times heard in peritonitis, as in perihepatitis in the right hypochondrium, or in the left hypochondrium with perisplenitis.

The venous hum or aneurismal bruit of abdominal aortic aneurism can be appreciated, or occasionally a venous hum over the liver from pressure on the vena cava. If pregnancy is present, there are the fetal heart sounds. The sounds over the stomach are of little diagnostic value, except the duration of the swallowing sound.

TOPOGRAPHIC ANATOMY

The position and relations of the stomach and intestines have been described, but for the purpose of physical diagnosis we must briefly refer to the normal relations of the other abdominal viscera.

In Fig. 21 are depicted diagrammatically the relations of the organs on the anterior surface of the body, and in Fig. 22 the relations on the posterior surface.

The Liver.—The general shape is that of a wedge with its base in the right hypochondrium, the upper surface lying in relation to the vault of the diaphragm, and its lower surface with the stomach, duodenum, gall-bladder, transverse colon, and small intestines; its anterior, lateral, and posterior portions are in relation with the abdominal parietes and lower right ribs.

Delimitation of the Normal Liver.—Mark a point 1 (Fig. 23) at the lower border of the fifth rib, between the left parasternal and mammillary lines, or about 2 to 2½ inches from the left edge of the sternum.

Point 2 lies in the fourth right intercostal space in the mammillary line. From 2 to 1 draw a connecting line, slightly convex upward on the right half and concave on the left, curving down at the lowest point to the base of the ensiform appendix.

From 2 draw a line nearly horizontally to the right and posteriorly, which should cut the midaxillary line in the seventh space and the scapular line in the ninth space, to the midspinal line. This line, front, side, and back, corresponds to the upper border of the liver.

To delimit the lower border, mark point 4 in the median line (linea alba) about a hand-breadth (3½ to 4 inches) below the base of the ensiform process of the sternum.

Mark point 3 at the lower edge of the ninth right costal cartilage, and another point 5 at the edge of the left costal arch on a level with the lower border of the sixth rib. A line should then be drawn from 3 to 4 upward and to the left. At 4 is indicated the notch between the liver lobes. From 4 a slightly curved line to 1, passing through 5, should next be drawn. The line from 3 to 1 indicates the lower anterior border of the organ.

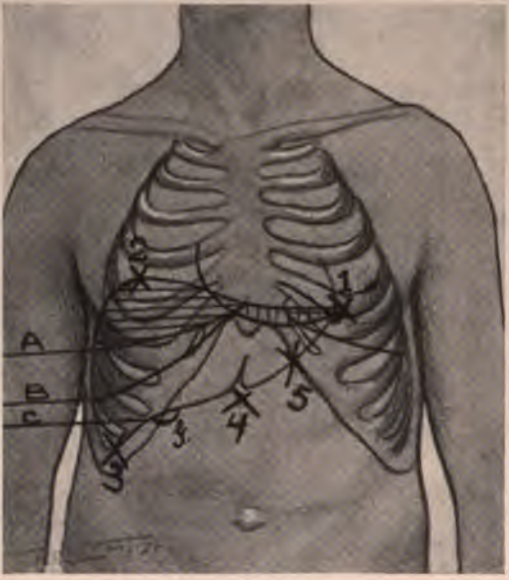

Fig. 23.—Delimitation of the normal liver (diagrammatic): *G*, Gall-bladder.

From point 3 draw a line backward and to the right, cutting the tenth intercostal space in the midaxillary line, from which point it joins the spine at a level of the eleventh rib (Fig. 24). This line demarks the lower border of the liver laterally and posteriorly, and is a continuation of the anterior inferior border.

This illustration shows diagrammatically the relations of the lower borders of the lung, pleura, and liver in the midaxillary line.

The left lobe lies to the left of the linea alba and extends nearly to the nipple, the notch lying in the midline. In the right mammillary line the liver extends from just below the level of the nipple to the costal margin.

The horizontal shadings in Fig. 23 show the portion of liver overlapped by lung, and the vertical shadings, that overlapped by the heart.

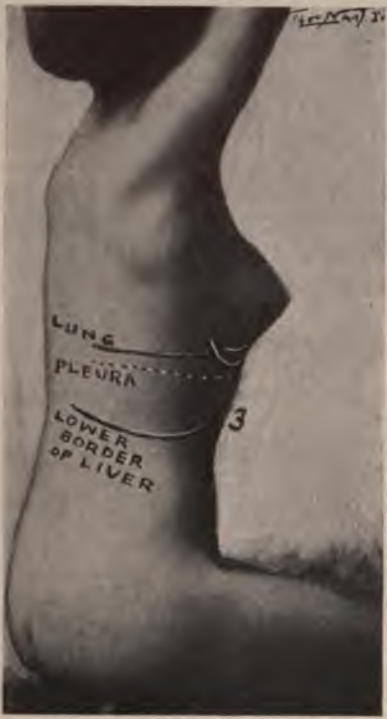

Fig. 24.—Lower border of liver (midaxillary line).

The gall-bladder, which is pear shaped, lies just internal to the ninth right costal cartilage.

PHYSICAL EXAMINATION OF THE LIVER AND GALL-BLADDER

Inspection.—The method advocated by M. Knapp I have found most practical. The patient stands with the abdomen uncovered facing a good light, the examiner slightly to the patient's right, so as not to interfere with the exposure to light. The lower edge of the enlarged liver shows on the abdomen as a linear transverse shadow, moving up and down with respiration and being especially prominent at the close of expiration, when there is a sudden check to the movement. Pulsation can occasionally be observed. Extreme enlargement may cause fulness in the right hypochondrium.

An enlarged gall-bladder can be appreciated in the same manner. Inspection may also be made with the patient in the dorsal position, the examiner stooping so as to bring the eyes on a level with the abdomen.

Palpation.—The head and shoulders should be slightly raised and the knees and thighs flexed, the patient being in the dorsal position. The examiner, sitting on the right side of the patient, should lay the right hand flat on the abdomen below the right costal arch, the fingers pointing upward and obliquely inward just to the right of the right rectus. Depress the fingers and feel for the resistant edge of the liver. The patient should be directed to take deep respirations, and by pressing inward and upward with the fingers the edge of the organ can be felt to move up and down.

As the liver may be enlarged, palpation for its lower edge must be begun from the level of the umbilicus upward. It is often necessary to feel for the notch of the gall-bladder or round ligament to determine whether it is the edge of the liver which is felt on palpation. One should note whether the edge is sharp or thick, or smooth or irregular. In some cases with thick abdominal walls the liver may be "spilled" against the latter by turning the patient on the right side, as in Fig. 25, thus rendering palpation more easy. The hand in this case may be in the reversed position.

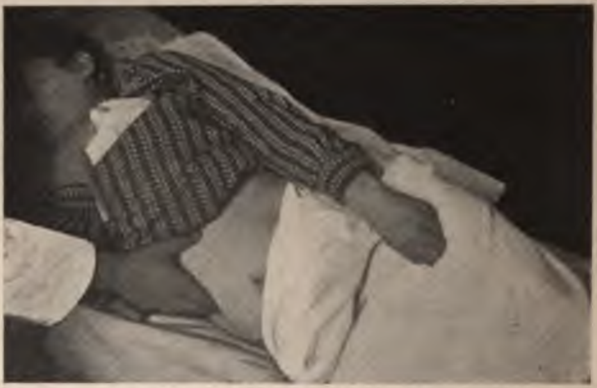

Fig. 25.—Spilling the liver.

The surface of the liver should be palpated, the left lobe in the epigastrium and the portion projecting below the ribs, if it be enlarged.

One should observe whether it is rough, smooth, nodular, or whether large tumor-like masses are present; also whether it is hard or soft and fluctuating; or if there is a thrill (hydatid), or friction during respiration, or pulsation. If the abdomen is distended the "dipping" method of palpation may be necessary.

The *empty gall-bladder* is not palpable. If distended it feels like a smooth pear-shaped tumor, moves with respiration, and is movable laterally, unless there are adhesions.

If malignant growth is present, the gall-bladder is irregular and nodular. If there are many gall-stones, Hutchinson describes the feel as of a "bag of nuts."

Percussion of Liver and Gall-bladder.—For anterior and lateral percussion the patient should lie down; for percussion posteriorly he should be sitting or standing.

The upper part of the right lobe is overlapped by the lung, and of the left lobe, a small area is covered by the left lung and heart.

Percussion over the covered part gives impaired pulmonary resonance, or modified dulness (deep, relative, or covered hepatic dulness). The part in contact with the parietes gives absolute dulness (superficial or exposed dulness).

It is necessary to delimit the entire area. Percuss downward, first in the mammillary line, beginning at the second intercostal space; then in the midaxillary line from the fourth interspace, and in the scapular line from the angle of the scapula. Percussion should then be made from below upward, in the midline from the umbilicus and from lateral and posterior points below the ribs.

Covered Hepatic Dulness.—Strong percussion should be employed, commencing above in the areas noted, and watching for the change from pure pulmonary resonance to impaired resonance, which denotes the presence of the liver. Impaired resonance is found normally in the fourth space in the mammillary line; in the seventh space in the midaxillary line; and in the ninth space in the scapular line.

Upper Limit of Exposed (Absolute) Hepatic Dulness.—Gentle percussion should then be employed and normally absolute liver dulness appears in the mammillary line at the sixth rib; midaxillary line at the eighth rib; and in the scapular line at the tenth rib.

Lower Limit of Hepatic Dulness.—Gentle percussion along the lines previously indicated from below upward will differentiate between tympanites and hepatic dulness. The lower limit normally is in the median line anteriorly a hand-breadth (3½ to 4 inches) below the ensiform; in the mammillary line, the tenth space. In the scapular line, it joins the dulness of the right kidney.

The vertical width of liver dulness is normally in the mammillary line 4 inches; in the midaxillary, 6 inches; in the scapular, 3 inches.

Percussion of the Gall-bladder.—This is only possible when it is distended or enlarged, in which event there is an area of dulness projecting downward and inward from the lower border of the liver and continuous with the dulness of the latter. In some cases the transverse colon may pass over the neck of the distended gall-bladder and separate its dulness from that of the liver by a tympanitic area. This is important to remember.

Auscultatory Percussion of the Liver.—The stethoscope should be placed over the middle of the area of the liver anteriorly, laterally, and posteriorly, and percussion be carried out on the lines already described. As a rule, simple percussion is sufficient; but the auscultatory method is of special value to determine whether a tumor is connected with the liver or not.

Thus in Fig. 26 the stethoscope is placed over point *S*. The note over the tumor *T* resembles more closely in intensity and quality the percussion note over the liver at *C* than it does over the point *A*.

General enlargement of the liver may be due to passive congestion usually from valvular disease of the heart, amyloid disease, cancer fatty infiltration, hypertrophic cirrhosis, leukemia, abscess, gumma or, rarely, Weil's disease.

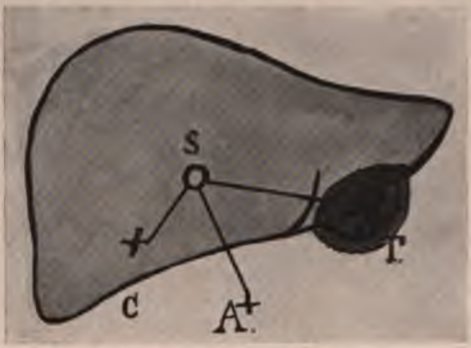

Fig. 26.—Tumor of liver.

Circumscribed enlargement of the liver, *i. e.* of the left lobe, is usuall due to abscess, hydatic cyst, gumma, or cancer.

Downward displace ment of the liver is causec by various intrathoraci lesions, such as emphy sema, effusions, etc., b subphrenic abscess, or a a part of a general ptosis o the viscera. In downwar displacement the upper surface, especially of the left lobe, is readil accessible to palpation and presents a rounded surface.

A prolapsed liver does not move as freely with respiration, o account of its separation from the diaphragm.

The consistence of the liver is somewhat diagnostic. It i abnormally hard, dense, and resistant in cirrhosis, carcinom amyloid disease, or syphilis.

A fluctuating swelling at the lower border may be a distende gall-bladder, abscess, or hydatid cyst.

The surface is smooth in fatty infiltration or degeneration, i passive congestion, and in amyloid disease; it is rough in tubercul peritonitis and granular to the feel in cirrhosis.

Hard nodules, varying in size, are suggestive of cancer; smoot elevated prominences occur with gummata. A smooth projectio may be due to abscess or cyst.

Topography of the Pancreas.—The pancreas (Fig. 27) li about 3 inches above the umbilicus, midway between the navel an the ensiform appendix, corresponding to the level of the secon lumbar vertebra.

It is about 6 inches long and lies deep in the epigastrium, tran versely across the spine, with its head resting in the curve of t duodenum and its tail extending to the spleen. The stomach cove it in front. It is rarely accessible to direct examination. The hea of the organ lies in close relation to the inferior vena cava, port vein, and common bile-duct, which are posterior. A cancer growth of the head of the pancreas may press upon these bloo vessels and cause edema and ascites, or upon the bile-duct and produce persistent jaundice.

Pain, fatty diarrhea, ascites, glycosuria, and jaundice may result from pancreatic disease.

Physical Examination.—Normally the pancreas cannot be palpated unless the patient is extremely emaciated.

An important physical sign of pancreatic disease is the *presence of a tumor* in the median portion of the epigastrium, midway between the navel and ensiform process. It is deep seated and often nothing more *than a sense of resistance* can be appreciated by the palpating hand.

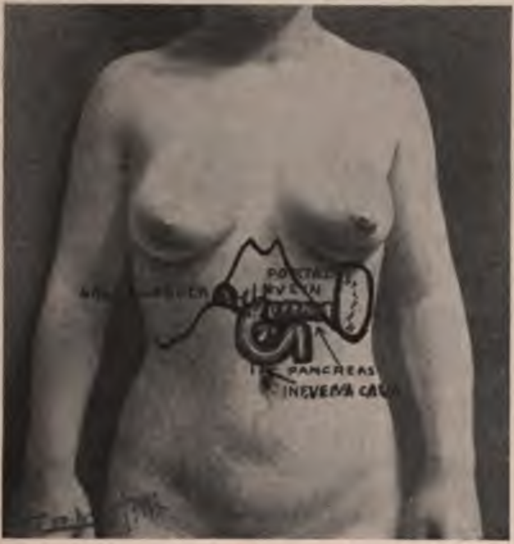

Fig. 27.—The pancreas.

The diseases in which these conditions are present are acute hemorrhagic or suppurative pancreatitis, chronic pancreatitis, and tumor—either carcinomatous or cystic.

Topography of the Spleen.—The spleen is oval, flattened in shape, and lies in the left hypochondriac region, measuring on an average 5 by 3 inches. It reaches from a point 1½ inches from the midspinal line posteriorly nearly to the midaxillary line, lying along the ninth, tenth, and eleventh ribs, the long axis being parallel with the ribs and running obliquely forward and downward, as in Fig. 30.

The lower two-thirds of its outer surface lie against the lateral abdominal wall, and the upper third is overlapped by the diaphragm, which separates it from the lower border of the left lung. The diaphragm lies above and the left kidney posteriorly, and it is in contact elsewhere with the stomach, pancreas, colon, and small intestine.

The anterior border is sharp and indented by two to four n

Physical Examination of the Spleen.—*Inspection.*— spleen is greatly enlarged, it may be visible as a protuberance ing from the left hypochondrium downward and inward, with respiration.

Palpation.—With the patient in the dorsal position a knees and thighs flexed, the examiner on the right side of should lay the right hand flat on the abdomen and with the tips exert pressure, pushing obliquely upward under the lef margin at the tenth cartilage. The edge of the enlarged can, as a rule, then be felt.

If it is not palpable, then request the patient to take deep when the sharp edge of the organ, which is smooth and notched, moving with respiration, will be felt riding over the tips and directed downward and inward.

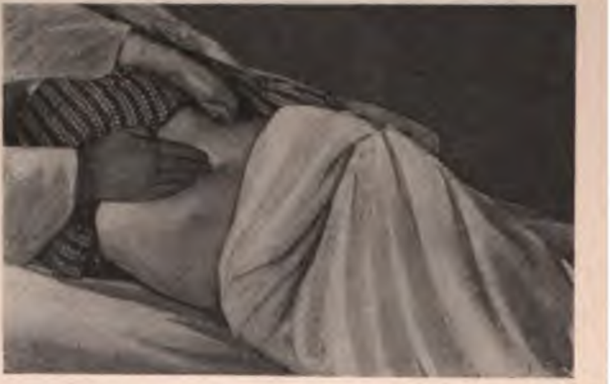

Fig. 28.—Examination of the spleen. "Spilling the spleen."

The left hand may be placed posteriorly between the end tenth and eleventh ribs and firm pressure be made, so as to organ forward and thus make palpation more easy. Norm spleen cannot be felt.

If the organ is enlarged a depression or space, into wh finger-tips can be sunk, can be detected at the posterior b the enlarged spleen between it and the erector spinæ.

An excellent method of palpation of the spleen is shown in By turning the patient on the right side, combined with p pressure, palpation is rendered easier.

It is often necessary to differentiate between tumor of kidney or spleen.

The spleen is oval, moves with respiration, is notched, has edge, a gap is present between it and the lumbar muscles, an no tympanitic resonance over it.

The kidney is reniform in shape and rounded, has no sharp edge or notch, and is overlaid by tympanitic resonance (Fig. 29).

Percussion of the Spleen.—The patient may be recumbent, partially turned to the right, midway between the dorsal and right lateral position, with the left arm extended over the head; or the examination may be made in the sitting posture. Percussion should be light, except over the posterior portion near the kidney.

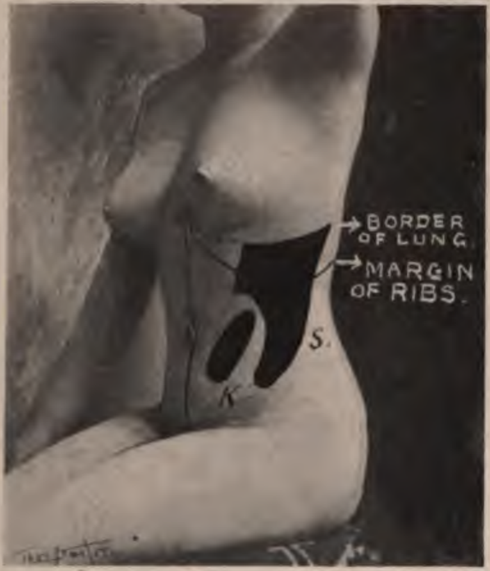

Fig. 29.—*S*, Tumor of spleen; *K*, tumor of kidney. Kidney tumor overlaid by tympanitic colon.

It should be carried out along the lines *A*, *B*, *C*, *D* in Fig. 30. Anterior percussion at the costal margin along the tenth rib, along *A* until the tympanites of the stomach is replaced by dulness usually at midaxillary line. From above percuss along *B*, commencing at the level of the angle of the scapula midway between the posterior axillary and scapular lines, passing vertically downward until pulmonary resonance is impaired, generally at ninth rib.

Percuss from below upward along line *C*, commencing below the border of the ribs, in or slightly posterior to the posterior axillary line, and passing upward until tympanites becomes dull, usually at eleventh rib.

Posteriorly percuss strongly from midspinal line at level of tenth rib and along the latter.

Splenic dulness should commence 1½ inches from the median spinal line. It is difficult to determine. The area of splenic dulness is oval, 2 to 2½ inches by 3 to 3½ inches. Dulness of over 3½ inches, on vertical percussion, shows enlargement.

Pleuretic effusion, consolidation at the left base, and fecal accumulation in the splenic flexure, may obscure percussion of the spleen. Palpation is the most important method and the most accurate.

Acute enlargement of the spleen occurs with infectious diseases, such as typhoid, malaria, etc., and in septic processes; chronic enlargement, with leukemia, malaria, cirrhosis of the liver, amyloid disease, pernicious anemia, etc. The organ may be displaced downward by intrathoracic pressure.

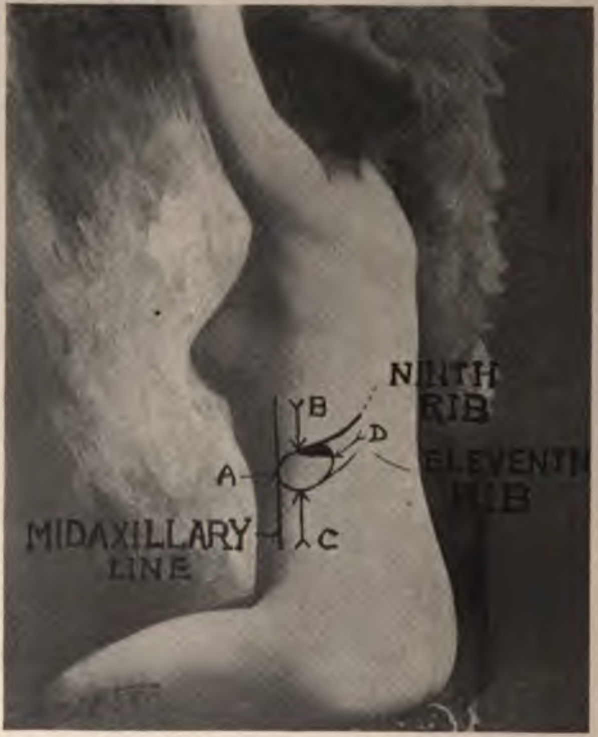

Fig. 30.—Lines for percussion of spleen.

Abscess, carcinoma, or hydatids may cause an unequal enlargement. Liver and spleen may be enlarged together in passive congestion, cirrhosis of the liver, leukemia, and in amyloid disease.

A floating spleen may occur as a result of congenital laxity of its ligaments or to overstretching from the increased size or weight. It may be part of a visceroptosis, usually in women. It is recognized by its mobility, shape, sharp edge, and notches.

Topography of the Kidneys.—The two kidneys lie against the posterior abdominal wall, one on each side of the spinal column, in beds of fat and connective tissue. They are of reniform shape. The upper end of the right kidney is in contact with the liver, and the left kidney with the spleen. They are retroperitoneal, the ascending and descending colon respectively lying in relation in front. The right kidney lies about ½ inch lower than the left. Each organ is about 4 inches long, 2 to 2½ inches in breadth, and 1 inch thick.

Surface Relations of the Kidneys.—Draw a horizontal line through the upper margin of the umbilicus; prolong the mammillary lines on each side downward until they intersect this horizontal line. The points of intersection lie about 3 inches on each side of the median line.

From the intersections measure upward 1 inch on the right and 1½ inches on the left mammillary line, and draw on each side a short horizontal line. The lower ends of the kidneys lie at these levels (Fig. 31).

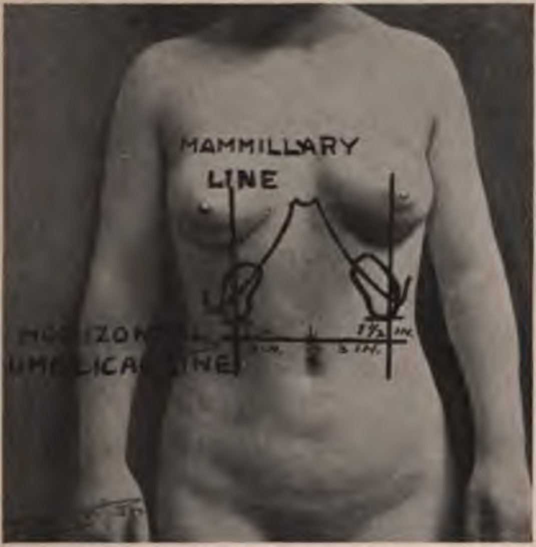

Fig. 31.—Anterior surface of kidneys.

One can measure 3 inches on each side along the horizontal line and then upward, without drawing the mammillary line. The kidneys extend upward and inward about 4 inches, one-third to the outer side and two-thirds to the inner side of the vertical lines.

Posterior Surface Relations of the Kidneys.—Draw a horizontal line across the back at the level of the tip of the spine of the eleventh dorsal vertebra; a second line at a level of the tip of the spine of the third lumbar vertebra. On each side draw a vertical line from the upper to the lower horizontal lines, 1 inch from the median line of the spine, and second vertical lines 2¾ inches away from the first vertical lines. Within these outer parallelograms lie the kidneys (Fig. 32).

The lower ends of these organs lie from 1 to 1½ inches above th iliac crests, the right ½ inch lower than the left. About a third of th upper ends are covered by the eleventh and twelfth ribs; the live overlaps the right kidney and the spleen the left.

Physical Examination of the Kidneys.—Inspection is seldo of service; palpation is most valuable; percussion is often uncertai

Inspection.—A large tumor of the kidneys may be visible in t anterior lumbar regions, extending into the umbilical region, wit outward bulging of the ribs on the affected side, such as in the ca

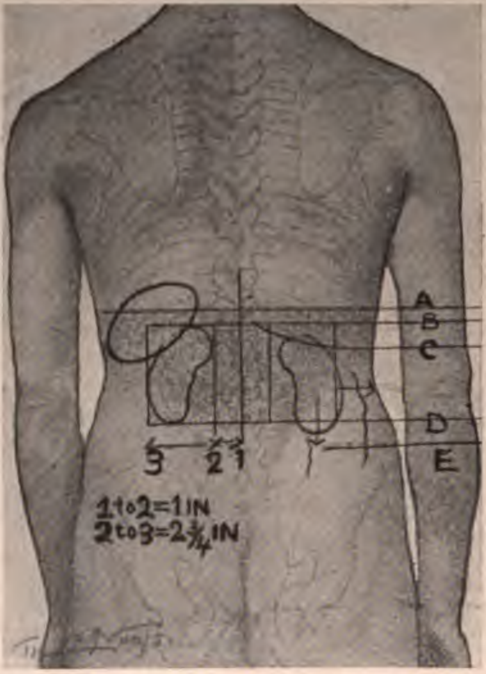

Fig. 32.—Posterior surface relations of kidneys: *A*, Lower border of lun *B*, level of spine of eleventh dorsal vertebra; *C*, lower border of liver; *D*, le of spine of third lumbar vertebra; *E*, colon.

of sarcoma, hydronephrosis, or cyst. A perinephritic abscess m become visible as a swelling in the posterior lumbar region.

Palpation.—This is most important. There are several metho described, of which the two following are the most practical:

Method 1.—The patient lies in the dorsal position, with the kne and thighs flexed, and the head is slightly raised to secure perfe relaxation. If the right kidney is to be examined, the left hand slipped under the back, so that it rests on the two lower ribs and t lumbar space below them. The right hand is laid flat on the abdom in front, resting below the costal margin to the outer side of t rectus, as in Fig. 33.

The patient should take deep and slow respirations, and during expiration firm pressure should be made with the fingers in front against posterior counterpressure, so that the kidney may be grasped between the hands.

If the kidney is normal in position and size, the extreme lower edge can be felt if the abdominal walls are not too thick. If the lower quadrant of the organ can be clearly palpated, it may be considered a movable kidney (of the first degree).

Various classifications of mobility have been given, some considering the organ when palpable in half its extent, mobility of second degree; entirely palpable, mobility of third degree; and when descending into the abdominal cavity, mobility of fourth degree. Others consider it movable if the entire length be accessible, especially if it can slip down as far as the horizontal umbilical line; and if it can be displaced below this or across the abdomen, as a floating kidney.

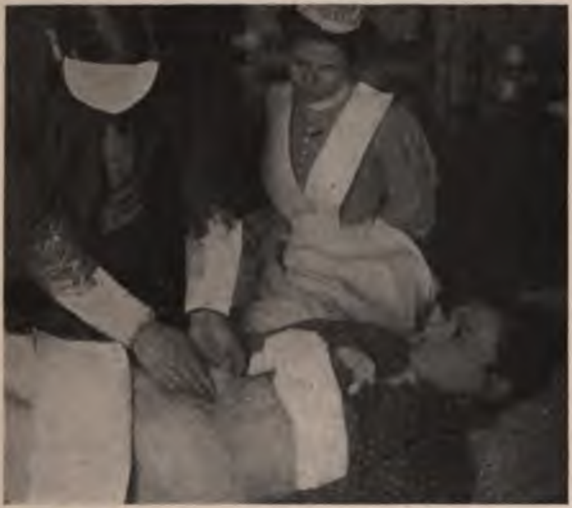

Fig. 33.—Palpation of kidney (older method).

I believe that any kidney which can be palpated to one-fourth of its extent should be considered movable, and the subdivision into various degrees of mobility to be excellent. A movable kidney from strain or traumatism is comparatively rare. Occasionally we find a congenital floating kidney with no visceroptosis. As a rule, I believe that in about 95 per cent. of cases of movable kidney the condition is merely part of a ptosis of the abdominal organs, and it may be considered to be practically pathognomonic of this condition. The right kidney is most frequently movable, though both may be so.

If the kidney is not found at its normal site, it should be searched for in the abdomen.

In examination of the left kidney, the position of the hands reversed.

Method 2.—This is, from personal experience, the most practi method of palpation, and the varying degrees of mobility can detected with greater ease than by the method described.

For examination of the right kidney, the patient sits up in b and the left hand of the examiner grasps the right flank, the thu resting under the costal margin, the fingers posteriorly. The pati breathes deeply, or coughs, or bears down, and the kidney can felt to slip down between the thumb and fingers, like "a pea i pod," or the lower margin or part of the kidney may be felt (Fig. 3

If the organ slips beyond the thumb and fingers or is not fou in its normal position, then the left hand grasps the flank m firmly and upward palpation is made with the right hand, as in Fig. 35, the patient gradually assuming the semi-oblique and, finally, the dorsal position, the final step, as shown in Fig. 36.

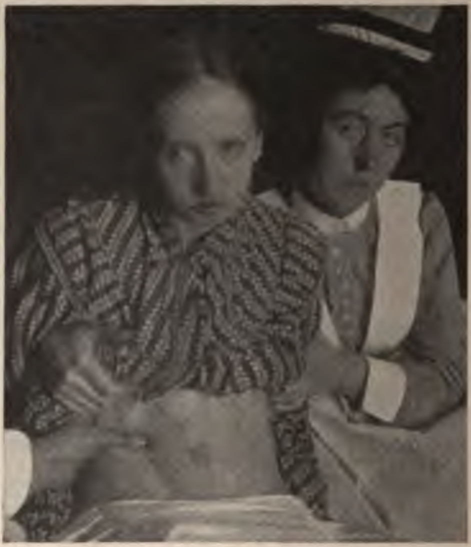

Fig. 34.—Palpation of kidney. Step 1.

A similar method with the patient standing is employed by Goelet, but the technic described seems preferable.

For palpation of the left kidney, the position of the hands is reversed, the right hand grasping the loin and the left hand anteriorly.

Tumors, hydronephrosis, and cysts of the kidney may be detected by palpation of the abdomen, as already described.

Brewer's Point.—Tenderness at the costovertebral angle is diagnostic of an acute inflammatory condition of the kidney, such as infarctions, etc., and aids in differentiation between this condition, appendix adherent to the liver, and acute cholecystitis.

Percussion of the Kidney.—The lower and part of the outer border of the kidney may at times be determined by percussion, comparing the dulness with the tympanitic note of the colon which lies anteriorly. As a rule, the thickness of the muscles, peritoneal fat, or fecal accumulation render the results uncertain.

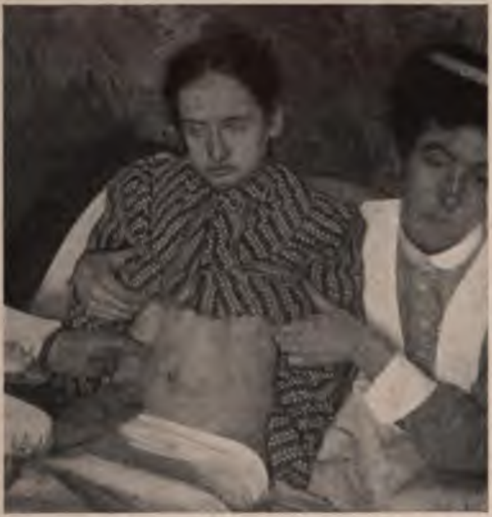

Fig. 35.—Palpation of kidney. Step 2. Patient in semi-oblique posture.

Position for Percussion.—The patient may lie on the abdomen, with one or two pillows placed underneath, to arch the back; or on the side, midway between the lateral and prone position, the physician sitting facing the patient's back and percussing the uppermost kidney.

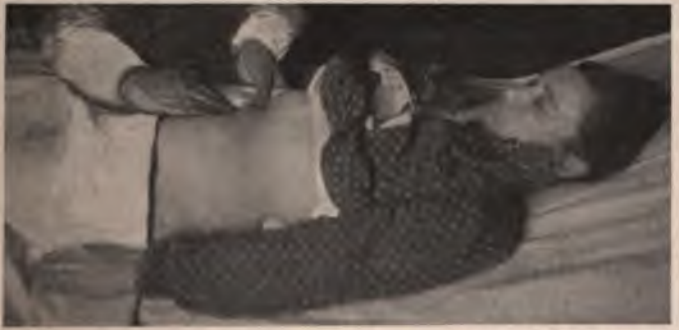

Fig. 36.—Palpation of kidney. Step 3 (final). Patient in dorsal position.

One should commence percussion in the middle of the area in which the kidney lies (Fig. 32), using heavy strokes, and percuss outward until the kidney dulness is replaced by tympanites; also percuss downward in the same way. Increased dulness shows enlarged kidney.

To Differentiate a Movable Right Kidney from the Distended Gall-bladder.—The kidney is movable in all directions, can be carried downward, does not move with respiration. Tympanites is found between it and the costal margin, the shape is reniform, and it may be pushed back into its normal position.

The distended gall-bladder moves with respiration, can only be moved laterally; if pushed away from the abdominal wall, it tends to resume its original position; and there is no tympanites between it and the liver dulness.

The chief causes of enlarged kidney are pyonephrosis, perinephritic abscess, hydronephrosis, cyst, echinoccoccus, carcinoma, and sarcoma.

An enlarged kidney tends to develop toward the front; an abscess, posteriorly, between the last rib and the iliac crest.

With enlarged kidney, the colon overlays in front of the tumor. With splenic tumor, this does not occur (Fig. 29).

If in doubt, inflate the descending colon with air through a colon-tube or catheter.

PART II

DISEASES OF THE STOMACH

CHAPTER V

METHODS OF PHYSICAL EXAMINATION OF THE STOMACH

General Considerations.—For an intelligent understanding of the subject, it is necessary to briefly define those conditions which constitute an abnormality in the position of the stomach and to differentiate between them.

The greater curvature of the normally distended stomach lies about 2 to 3 fingers-breadth (1½–2¼ inches) above the umbilicus. The normal position of the organ has been indicated in Chapter I. It must be remembered that some possess an abnormally large stomach; and that it can only be considered to be dilated if there are symptoms associated which point to this organ.

If there is atony of the stomach, with motor insufficiency, the patient having gastric symptoms, while the *lower border of the stomach is defined* at the level of the umbilicus or below it, the lesser curvature maintaining its relations to the diaphragm, we may consider the organ to be dilated. This constitutes the atonic type of dilatation, which is extremely common. Many of this class suffer from autointoxication with nervous sequelæ, and are found in great numbers in our asylums and among our nervous cases. Attention may be diverted from the gastric symptoms and they may even be slight.

As a result of pyloric spasm, or benign or malignant stricture of the pylorus, or any obstruction in the pyloric region interfering with the egress of the gastric contents, we have the so-called stenotic type of dilatation of the stomach.

In these cases the lesser curvature retains its relation to the diaphragm, while the lower border extends to the umbilicus or below it, and gastric symptoms are present to a marked degree and of a special type.

With gastroptosis (prolapse of the stomach) the suspensory ligaments of the stomach are relaxed and the entire organ sinks, *the lesser curvature as well as the greater.* In aggravated cases the lesser curvature looks inward to the right, and the greater curvature

...val to the left. The pylorus may often lie below the level of umbilicus. With this condition, movable kidney—especially of the right organ—is present, and may practically be considered pathognomonic in my opinion, as only in about 5 per cent. or less of cases is mobility due to strain or injury. The congenital floating kidney, without ptosis of other viscera, is excessively rare.

There may be various degrees of gastroptosis, a loop-shape, or even a vertical stomach similar to that of the fetal period, which are pictured in the chapter on Gastroptosis. *The change in position of the lesser curvature is*, therefore, *diagnostic of gastroptosis*, and not the position of the lower border of the stomach. *The presence of movable kidney is also diagnostic.*

It seems advisable to describe the methods generally employed, and at the end of the chapter to briefly summarize those which are of most practical value.

Preparation of the Patient.—On the day or night previous to examination, the bowels should, if possible, be thoroughly emptied by a cathartic. If there is much tympanites, it should be relieved by a hot enema of about 1 liter (1 quart) of normal saline solution or soapsuds and water; or, if the condition is marked, then by enteroclysis at 110° to 120° F. with a recurrent tube (Kemp's) or two catheters. This carries off the gas in a satisfactory manner.

The patient should be examined in the dorsal, semi-oblique, and standing positions.

Inspection.—Examination in the dorsal position should first be made.

A recognizable bulging, distinct from the epigastrium, especially if it occur in the umbilical or hypogastric region, may be due to a dilated stomach; the epigastrium under these conditions is usually hollow and depressed. Inspection is often of assistance in thin patients, especially after artificial distention of the stomach with carbonic acid gas.

Peristaltic movements of the dilated stomach are at times observed.

Kussmaul has noted very active peristaltic movements in the dilated stomach (peristaltic unrest), the waves passing from the linea alba below the umbilicus in an upward direction and to the right to the lower margin of the liver. This is found present in cases in which stenosis of the pylorus exists.

We can facilitate inspection by placing the patient upon a raised table, the head toward the window, the shades being arranged so that the light enters on a plane only slightly above that of the patient, and is directed from the head toward the feet. The examiner, standing toward the foot of the table and bending from side to side, can at times make out shadows cast by the inequalities of the abdomen. The shadows move with respiration. By this method the size, shape, and position of the stomach can often be made out.

Knapp places the patient in the same position, but stands at the side or at the shoulders, and brings his eyes down to the level of the abdomen and observes the respiratory waves passing over its surface. After some experience one can detect delicate transverse lines or waves passing upward and downward with respiration. These lines correspond to the curvatures of the stomach.

More recently he places the patient facing a good light, and, standing slightly to the side, observes the movements of the transverse lines. I have seen good results from this method, especially for determination of the lower border of the stomach.

The following signs I have found quite reliable: With the patient in the recumbent position, a marked concavity between the costal arches—extending from the ensiform process to or below the umbilicus, with a vertical median sulcus, wider above than below, the abdomen being flattened in the central part and bulging in the lateral regions—is significant of gastroptosis. In the erect position the epigastrium becomes still more depressed, while the umbilical and especially the pubic regions bulge outward. Tumors of the stomach may sometimes be observed, causing slight projection or protuberance on the abdominal wall.

Palpation of the Stomach.—Inspection should be supplemented by palpation. Palpation should be performed gently and the hands of the operator should be warm.

The patient should be in the dorsal position with the legs flexed, to relax the abdominal muscles. He should breathe naturally and keep the mouth open to aid relaxation. The physician should be seated on the right side of the bed and palpate with the right hand, which should be flat or slightly bent upon the abdomen, with the ulnar side down. One can stroke from above downward, and with practice it is possible, in some cases, to feel the stomach-wall and appreciate the position of the greater curvature, as the stomach gives a more uniform elastic sensation than do the intestinal walls. Some commence palpation from below and work upward, dipping in the ulnar edge of the hand rather deeply. By these means it is at times possible to determine the position of the greater curvature.

By palpation we can discover if nephroptosis is present. Diastasis of the recti muscles and floating tenth rib can also be determined These conditions are significant of gastroptosis. Under "Inspection" I have noted the signs that are significant of gastroptosis. If we find a "movable kidney," this renders our diagnosis conclusive.

By gentle palpation one can frequently discover a tumor, its position, size, consistency, and mobility. Occasionally, more pressure is necessary and the palpating hand may be reinforced by the other hand, after the method described.

Sensitive or tender points can be located by palpation; for example, the circumscribed tenderness of an ulcer or the diffus tenderness of the gastric region in acute inflammation. Boas ha devised an algesimeter for indicating the degree of pain. T. Kilme has also an instrument for the same purpose.

Considerable care should be exercised in palpation in cases c suspected ulcer, and I prefer the hand for this purpose.

Percussion of the Stomach.—The accurate determination c the position and size of the stomach is often difficult by simpl percussion. The sound varies, according to whether the organ i empty or filled with air, food, and water.

The position of the patient, whether lying down, semi-obliqu or standing, modifies the findings. In order to obtain results th stomach should contain some air. Dehio has demonstrated, bot on living subjects and on the cadaver, that *if the stomach is empt* the tympanitic sound which we produce on percussion is due to th colon and not to the stomach, since the latter is contracted into th left concavity of the diaphragm and is not in contact with the anteric thoracic wall. Hence the time at which the examination is mad is important. Moreover, the *lower curvature* tends to fall away fro the abdominal wall.

The patient should first be examined in the dorsal position wit the knees flexed.

This method determines with fair accuracy the upper right an upper left portions. The percussion hammer is sometimes an ai The absolute determination of the lower border by percussion more difficult. It is rendered easier if the bowels have been tho oughly emptied, since the colon is then less likely to ride over th greater curvature. The percussion sound over the colon is light and does not equal that over the stomach. The stomach sound of greater intensity and clearness and of higher pitch. This, course, refers to conditions when air is present as the factor. Foo or fecal contents alter the result, which is further modified by pe cussion in the semi-oblique and standing positions.

As a rule, there are some contents in the transverse colon, so th we have the tympanites of the stomach merging into dulness flatness. With gastroptosis, determination of the position of th organ by simple percussion is often difficult.

The presence of a tumor can frequently be determined by percu sion.

Auscultatory Percussion.—With this method we employ th stethoscope. The chest-piece may be placed above the seventh r in the left mammillary line, or between the tip of the ensiform proce and the left costal margin; or in the same vertical line, but slight below these points. First percuss near the stethescope to fix th characteristic sound. The tympanites of the stomach is transmitt generally through the liver and lung. The percussion should b begun *well distant* from the possible location of the stomach, an should be performed in the vertical direction, downward and upward, and also laterally. One should begin nearly at the symphysis and percuss in vertical lines upward (Fig. 37).

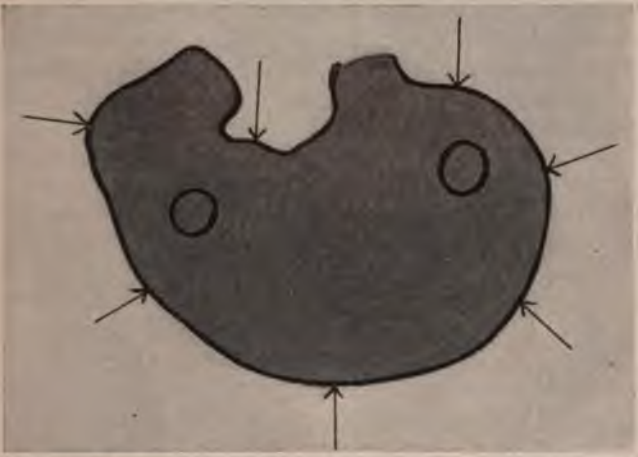

Fig. 37.—Vertical lines of auscultatory percussion. Circles show positions of stethoscope.

The patient should be in the usual position, as described, and should hold the stethoscope for the operator against the abdomen.

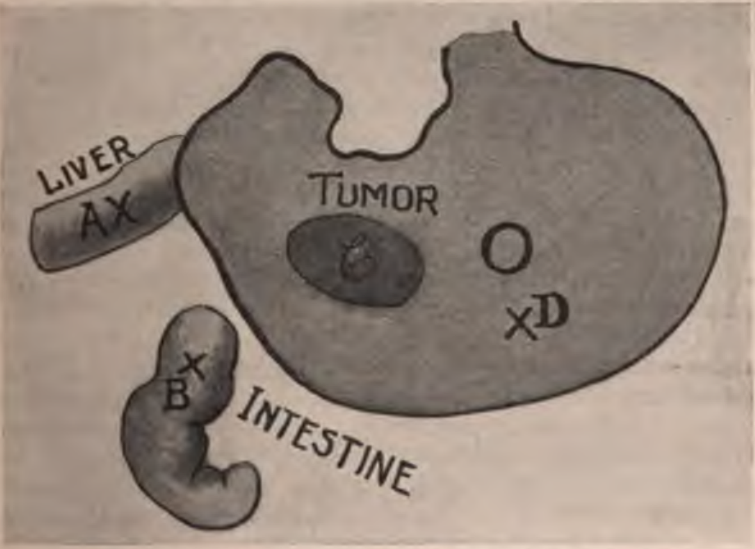

Fig. 38.—Auscultatory percussion of tumor of stomach wall.

A sound of greater intensity and clearness and of higher pitch denotes the border of the stomach. The greater bulk of the organ, when dilated or in a condition of ptosis, lies to the left of the median lin We must remember that some cases of marked dilatation extend great distance to the right of the abdomen.

This method is of value in determination of a tumor of th stomach.

Place the stethoscope at *O*, Fig. 38, and percuss toward the tum from all directions. The sound heard over the tumor (*C*) differs i character from that heard over the stomach at *D*. If the growt involves the stomach wall, *C* resembles *D* much more nearly tha *A* (percussion of liver) and B (percussion of intestine) resembles (stomach percussion).

Differential Diagnosis.—If a tumor lies at *D*, near the margi of the liver (Fig. 39), tumor of the liver is excluded as follows: pla stethoscope at *S*, over liver. Percussion note over *D* resembles no over *G* more than it resembles that over *F*. Then to *exclude intestin* shift stethoscope to *K*, over stomach. Percussion over *D* resembl that over *M* more closely than that over *G* resembles that over The tumor is, therefore, of the stomach.

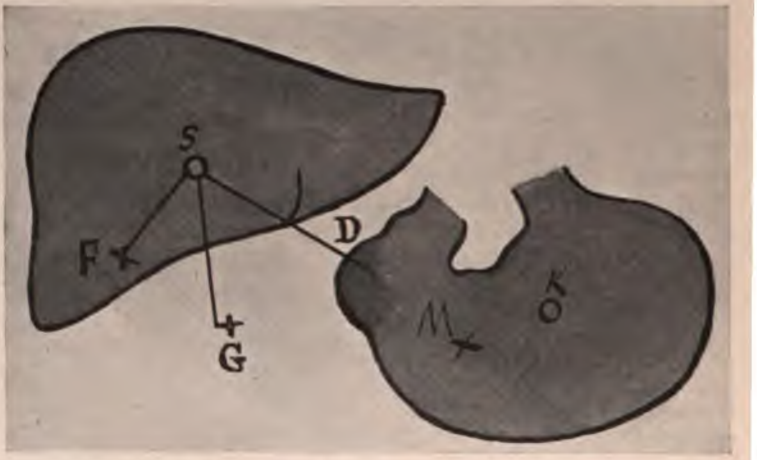

Fig. 39.—Differential diagnosis.

Reichmann's Rod.—This consists of a short ivory rod, wi circular grooves and intervening projections, like the handle of ivory knitting-needle. The rod is pushed firmly down over t stomach at a right angle to its surface (in a vertical line to t abdomen), and is gently stroked with the finger. The stethosco is applied over the organ and the "pitch" carefully observed. Wh the rod passes beyond the limits of the stomach, a change in "pitc occurs.

Scratch Method of Auscultatory Percussion.—This meth I have found satisfactory in many cases.

The stethoscope is placed on the abdomen below the left border of the ribs, and with the index-finger of the right hand the abdominal wall is scratched gently by the examiner so as to secure the "normal pitch" over the stomach. The abdomen is then scratched lightly from below upward, commencing below the umbilicus, and the *change in "pitch"* is readily observed when the *lower border of the stomach is reached.*

If gastroptosis is believed to exist, the stethoscope is placed more to the left, and the "scratch pitch" noted from left to right. The method is shown in Fig. 40. It is of special value in determining the lower border of the stomach.

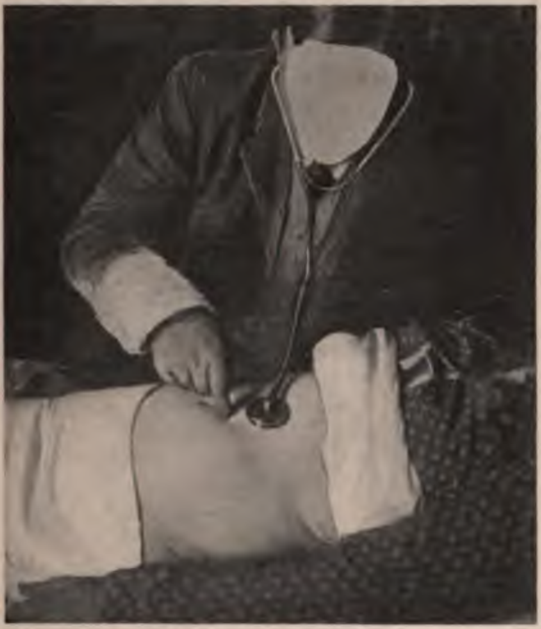

Fig. 40.—"Scratch method" of auscultatory percussion.

Flicking.—This method was described and illustrated in the previous chapter.

Inflation of the Stomach with Carbonic Acid Gas.—Carbonic acid gas inflation is employed to render the stomach visible to inspection; to aid the determination of the position of the lesser curvature, as well as the greater, and so enable a differential diagnosis between dilatation and gastroptosis.

The method is to administer, first, one-half glass of water in which about 1 dram of tartaric acid is dissolved and, after this one-half glass of water containing from 1 to 1½ drams of soda bicarbonate. If small quantities are employed the stomach will not become visible and palpable.

There are certain objections to this procedure. At times ther is considerable escape of gas through the cardiac orifice or pyloru and the small intestine may be distended. This is a possible source o error. There may be sudden hyperdistention of the stomach, wit resulting pressure on the heart and lungs, and unpleasant or eve dangerous symptoms result in the aged or in a patient suffering fro cardiac or pulmonary disease. In the chapter on Acute Dilatatio of the Stomach the effects of sudden distention of the stomach o the heart and circulation are described by Thomas Satterthwait and the author.

When there has been a hemorrhage, or symptoms of ulcer o cancer, or signs of peritonitis, the use of this method is contra-indicated. Several fatal accidents have occurred. It sometime irritates the mucous membrane. One could employ a stomach-tub and Dr. Rose's carbonic acid gas generating-bottle as a substitute Carbonic acid gas inflation is valuable in those who are in fai physical condition.

In the determination of the position of a tumor, whether it lie on the anterior or posterior surface of the stomach, it is of service. A posterior tumor will disappear under inflation. This is fully described under Cancer of the Stomach.

Inflation of the Stomach with Air.—This consists in introducing a soft stomach-tube and slowly pumping air into the stomach with a double-bulb or a Davidson's syringe. The tube should be introduced with the patient sitting up in bed, and he should then gently recline on the back, and inflation should be carried out. It possesses the advantage that the amount of air pumped into the stomach can definitely be regulated. Fill a vessel with 1 liter (1 quart) of water, invert it over a pail of water, and note how many compressions of the bulb displace the given quantity of fluid. One can thus estimate the quantity of air pumped in at each compression. The first few squeezes of the bulb should be given rapidly, so as to cause spasmodic closure of the pylorus.

The same indications and contra-indications exist as for the use of carbonic acid gas. There is the advantage of being able to regulate more definitely the degree of inflation. Some patients, however, object to the passage of the tube. If there are discomfort or unpleasant symptoms from either method of inflation, the condition should be immediately relieved by the passage of the stomach-tube.

Fürbringer suggests that when we inflate with air the tube should be introduced only to the middle of the esophagus, and air should then be pumped in. He claims that this procedure prevents retching.

Inflation of the Stomach with Water.—To Dehio we must give the credit of determining the position of the stomach by water inflation. He percusses over the patient's stomach, preferably with the organ empty and the patient in the erect position. He then administers a glass of water (8 ounces), not too cold, and percusses the area of dulness. He follows this with a second, third, and fourth glass of water, percussing each time, and notes the position and extent of the dulness. The patient is then directed to lie on his back and tympanites will appear where dulness previously existed. This conclusively demonstrates that the area corresponded to the stomach.

If there is pronounced dilatation or ptosis, a single glass of water will often cause dulness to appear below the navel or in the inguinal region. The results may be obscured in patients with much adipose tissue or if there is fecal accumulation in the colon. In this event, it should be cleared out by injection. I have also found the following method of value, especially if there be some gastric contents: first, place the patient in the *semi-oblique position* and percuss the stomach; then administer 2 or even 3 glasses of water. We secure stomach tympanites above, then a band of stomach dulness and intestinal tympanites below. It is easier to differentiate between dulness and tympanites than between two types of tympanites.

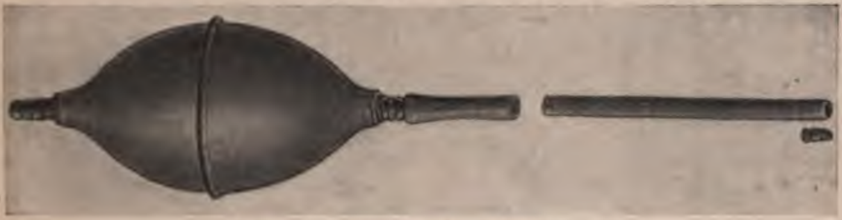

Fig. 41.—Kemp's stomach-whistle.

There are numerous complicated methods by means of inflatable bags, manometers, etc., for determining the position of the stomach, which are scarcely of practical value. Leube introduces a stiff sound and determines the position of its lower end through the abdominal walls. This method does not seem to be safe. Others differentiate between the stomach and the colon by inflating the colon with air or carbonic acid, employing the same methods as in the stomach, only using twice the quantity of soda bicarbonate and tartaric acid. Rose's apparatus would prove of value to inflate the bowel. To further differentiate, water was given by the stomach. Some first empty the bowel thoroughly and then inflate the intestine with water. It is often difficult for the patient to hold the enema.

There are two other methods for determining the lower margin of the stomach: First, the administration of small quantities of soda bicarbonate and tartaric acid, with the patient in the standing position. In some cases one can approximately map out the lower border of the stomach by listening to the "sizzling sounds" with the stethoscope. Second, the use of the stomach-whistle (Fig. 41). This consists of a rectal tube of small caliber, with a whistle in the end. To the other extremity is attached an ordinary stoma aspirating bulb without valves. The tube is inserted into stomach, the finger placed over the open end of the bulb, and a sin bulbful of air is forced into and aspirated out of the stomach by ra and short intermittent contractions. This entirely eliminates possible chance of distending the stomach with air and the org remains practically empty.

A stethoscope is placed over the abdomen and the point greatest intensity of sound is marked by a cross with a colored pen The tube is pushed in and out and the various points of sound marked—the lowest is in the lower border of the stomach. The can be applied in place of the stethoscope. Transillumination of t organ was then performed and the lower margins absolutely corr ponded. The method of administering water and then blowi air into the stomach through a tube, and producing "bubbli sounds," only gives the level of the fluid, and not accurately t lower margin of the stomach. The whistle will not differentia between dilatation and gastroptosis. This experiment with t stomach-whistle demonstrated that in the standing position t stomach, when empty, descends to the full length of its suspenso ligaments, and its lower border is at a constant level or within abo 1 inch of the same, whether the organ be full or empty. It was the same level when a pint or more of fluorescent fluid had be ingested as when demonstrated by transillumination.

Splashing Sounds.—The splashing sounds of the stomach a produced when water and air in the organ are agitated togeth when either the whole body or the stomach alone, is shaken. Th are best demonstrated by rapidly tapping with the index- and midd fingers of the right hand over the stomach several times in successi without removing the fingers, as in striking chords on the pian The patient should be in the dorsal position with the lower liml flexed.

The sounds resemble those produced by shaking a rubber b containing air and water.

They can be elicited in many people in ordinary good healt shortly after meals, but if found at an abnormal time or in an abno mal position, are of diagnostic value. If present an hour after test breakfast, the patient suffering from gastric symptoms and t position of the stomach being normal, they are significant of simp atony. This is true if they be found several hours after an ordinar meal or on an empty stomach. If the splash is present in an abnorm position at the level or below the umbilicus, it shows the lower bord of the stomach lies abnormally low, and that either dilatation gastroptosis is present.

The presence of movable kidney demonstrates it is a ptosi The upper border may be determined to be in an abnormal positio by inflation or gastrodiaphany, as a further test.

The splashing sound determines the position of the lower border of the stomach with greater accuracy than percussion. Some patients hold the abdomen rigid, so that it does not appear on examination, but it can be produced artificially for examination purposes.

The following is a simple method to differentiate between stomach and intestinal splash:

In Fig. 42, if the splash be found at *A*, mark same on abdominal wall; then give several glasses of water or, preferably, Vichy. If the splash at *A* be intensified, it is stomach splash. If, on the other hand, the splash appear at *B*, this is the *true* stomach splash and *A* is the intestinal splash.

If no splash is present on examination, it can be created artificially by giving several glasses of water or, preferably, Vichy. Sometimes 3 or 4 glasses are required; the patient takes several deep breaths, the splash being determined during expiration. I find the determination of the lower border of the stomach most accurate by means of the splashing sound.

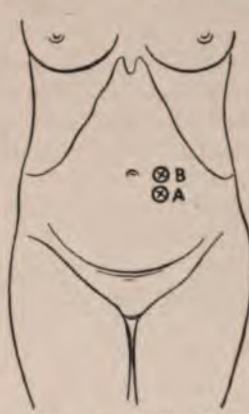

Fig. 42.—Splashing sound. Differential diagnosis between stomach and intestinal splash.

Deglutition Sounds.—These were first described by Kronicker and Meltzer.[1]

When drinking, a sound is heard simultaneously with the act of deglutition, which is termed the first deglutition sound. A second sound is noted about seven seconds later. Both sounds can be heard by placing the ear or stethoscope at the ensiform process. As a rule, only the second sound is heard. If the first sound is heard, the second may be present or absent. The presence of these sounds assists in forming judgment as to the permeability of the cardiac orifice. If they are absent, the ingested liquid has remained in the esophagus, and hence a tight stricture is present. If the second sound is markedly delayed, there is probably partial obstruction.

Other Sounds Have Been Described.—Dripping sounds, arising from the passage of fluid along the gastric wall, are suggested as a means of mapping out the stomach. The method is inaccurate.

The succussion sound, obtained by shaking the body of the patient, is not as accurate a method as by tapping.

Gurgling sounds occur from the contraction of the empty stomach about air or gas. Sounds are heard in the stomach due to movements imparted to the organ through the respiration, and also ringing sounds, imparted from the heart in gastric dilatation.[2]

These sounds are of no diagnostic value. Occasionally sizzling sounds are heard on auscultation, which are produced by fermentati of the gastric contents. They resemble the sounds produced af the administration of soda bicarbonate and tartaric acid, with t resulting generation of carbonic acid gas.

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[1] Centralb. f. die med. Wissensch., 1883, No. 1.

[2] Laker, Wiener med. Presse, 1889, Nos. 43 and 44.

Esophagoscopy.—Mikulicz, Rosenheim, Kelling, Von Hacke Einhorn,[2] and many others have advocated this method.

In the earlier instruments the source of illumination was outsi the esophageal tube. The instrument with the lamp at the end the tube near the point to be inspected, such as devised by Einho is more practical.

In Fig. 43 is depicted Einhorn's instrument, which is read understood.

The obturator is inserted and held in place by the plug *d* and t wires connected with the battery.

After introduction, the plug is removed, the obturator wit drawn, and the current turned on.

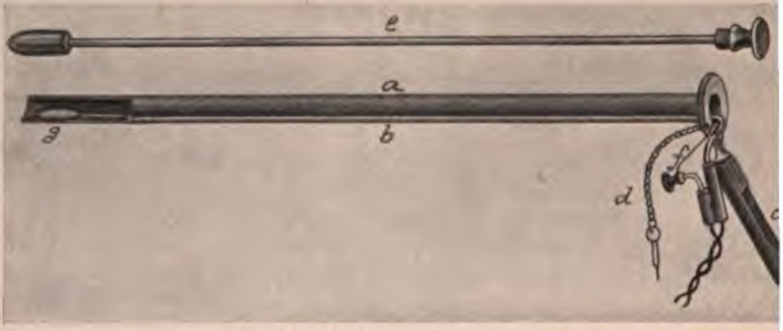

Fig. 43.—Einhorn's esophagoscope.

Technic.—The pharynx should be sprayed with cocain (5 p cent.). The patient sits on a chair with a straight back. T instrument with obturator inserted is immersed in warm water a inserted like a pen along the roof of the mouth to the posterior w of the pharynx, the head being thrown backward on the shoulde It is then pushed down in a vertical line into the esophagus witho the exertion of any force. By this method it is unnecessary to pre down on the tongue, and thus gagging is avoided.

The lips of the patient should not be pressed upon by the instr ment. The obturator is removed, the light turned on, and the e of the operator applied to the opening.

For complete inspection of the esophagus, the instrument gradually withdrawn.

This esophagoscope is made in several lengths and in two sizes.

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[1] Beiträge zur klinischen Chirur., Bd. 20, 1898, pp. 141, 275; Ibid., Bd. 1901, p. 128.

[2] New York Med. Journal, Dec. 11, 1897.

Indications.—Esophagoscopy is of chief value for the removal of foreign bodies. It may be employed in cases of suspected ulceration and for topical application thereto. In suspected cancer it should be used with caution.

Contra-indications.—Aneurism or recent hemorrhage.

Gastroscopy.—This method of examination of the gastric mucosa was inaugurated by Mikulicz in 1881.[1]

The general principle of the instrument is based on the cystoscope. Rosenheim has devised a new instrument, and Chevalier Jackson has carried on numerous investigations in gastroscopy. The latter has conclusively demonstrated that general anesthesia should be given for such an examination.

I must confess that in cases of suspected cancer or ulcer I would not submit a patient to the certain degree of risk from the introduction of an instrument of the present type; and in milder cases of gastric disturbances I can see no advantage, except for the purpose of scientific study. It would seem that a large number of normal organs, as well as mild types of gastric disease, should first be investigated as a basis for comparison, and that the instrument should be improved upon before the method can be generally recommended.

Gastrodiaphany or Transillumination of the Stomach.—Casenave, in 1845, first applied the method of transillumination to living tissues. In 1867 Milliot succeeded in transilluminating the stomachs of animals and experimented with the stomachs of cadavers, but to Max Einhorn, of New York, the credit is due of being the first to demonstrate transillumination of the stomach on the living subject and the practical value of gastrodiaphany. His instrument, which he denominates a gastrodiaphane, consists of a soft-rubber stomach-tube, at one end of which is fastened an Edison lamp. Conducting wires run through the tube to the battery, and there is a current interrupter at some distance from the tube. The lamp is enclosed in a glass bulb, to act as a reflector and prevent the action of heat on the stomach. He has the patient drink only 1 or 2 glasses of water, so as not to distend the stomach, inserts the light, and examines the case in a dark room, either in the sitting or in the recumbent position.

Heryng and Reichmann employ a modified tube, with a water cooler about the lamp. Kutner and Jacobson, under Ewald's direction, performed a great number of experiments.

These experimenters, together with Meltzing, are the chief foreign investigators with gastrodiaphany. Manges, Stockton, and many others have employed it. Among various gastrodiaphanes are those of Hemmeter, Lincoln, Solis-Cohen, Koplik, and Lockwood. To Lockwood we must credit a decided advance in the type of instrument—a fine, wire-bound cable (rubber insulated) and a small light, no larger than a 5-grain capsule. The cut of my instrument, the "circumscribing gastrodiaphane," will sufficiently explain the

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[1] Wiener med. Presse, 1881, No. 45.

Lockwood instrument, after which it is modeled, with cert additions.

The Circumscribing Gastrodiaphane.—A series of observati with transillumination of the stomach suggested an improveme on the gastrodiaphanes in use. Manipulation of the tube after electric light has entered the stomach frequently causes gagging a at times, vomiting, interfering thus with the accuracy of the meth The cables of all the instruments were found unsatisfactory in ca of gastroptosis of great degree when we endeavored to explore ca fully the pyloric end of the greatly dilated stomach. It was impos ble to guide the light in a definite direction; it would sometimes p to the right, sometimes to the left, and often it was necessary to dr it in and out a number of times for a distance of several inches.

The instrument I devised to overcome these drawbacks has cable about 6 inches longer than the Lockwood gastrodiaphane, a

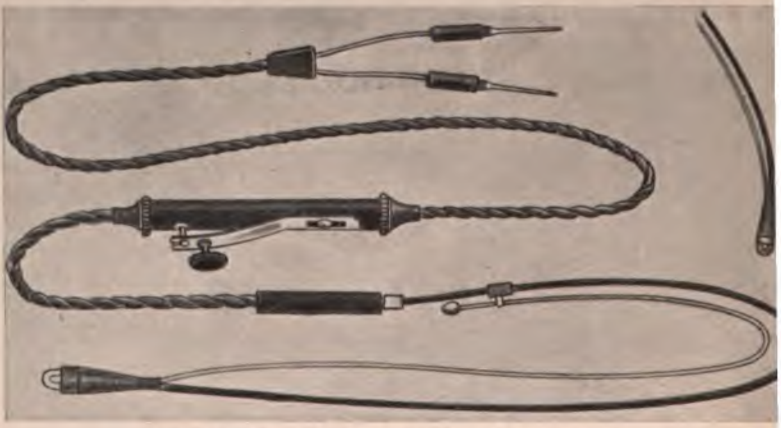

Fig. 44.—Kemp's gastrodiaphane (circumscribing).

is of about the same caliber (Fig. 44). The cable is more flexi for the space of ¼ inch at about the same distance from the light in effect, a joint at this point. At the base of the light is attach an extremely thin accessory cable, covered with rubber. This ru parallel with the main cable and increases the diameter only a ve slight degree. After introduction of the instrument the main ca is held firmly and the accessory cable drawn upon. By turning t cable at the same time, the instrument can be directed in the desir direction. By manipulation of the accessory cable the main ca can be so bent that the light will explore the entire wall of t stomach anteriorly, and can be made to pass up to the pylorus a along the borders of the ribs. The lesser curvature is thus explore

Care should be taken that the cables are parallel when pass into the stomach, and the accessory cable should be relaxed befo withdrawal. The main cable, except at the joint near the ligh is stiffer than the Lockwood light. Eight dry cells are employed with a rheostat. Wappler manufactures a small pocket battery with six cells, which I have found excellent. An extra lamp should be carried. Water was the medium formerly employed.

A great advance in the technic of gastrodiaphany is the employment of fluorescent media.[1]

There are three such media found to be of value:

Bisulphate of quinin, gr. 10 (0.6), in a pint of water. The addition of ♏5 (0.3) of dilute phosphoric acid intensifies its action. The same amount of dilute sulphuric acid may be substituted. The reaction of the quinin solution is acid and the fluorescence a very pale violet. Increased acidity intensifies its action and fluorescence disappears if the solution is rendered alkaline.

Esculin. This is derived from the Œsculus hippocastanum (horse-chestnut), indigenous to Europe; 15-grain doses have been used in malaria. One can employ small doses, gr. $\frac{1}{8}$ to $\frac{1}{2}$ (0.008–0.032), in a pint of alkaline solution, which gives a blue fluorescence. This preparation is difficult to secure and is expensive.

Fluorescein (phthalic anhydrid, 5 parts), a naphthalin product, and resorcin (7 parts), heated to 200 C. (392 F.). It is a reddish powder, faintly soluble in water, with a neutral action, and gives thus no fluorescence; soluble in alcohol and in alkaline media, it gives a green fluorescence like liquid opal. It has been employed to detect ulcers of the cornea. It can be secured from Merck & Co., and is extremely cheap.

The addition of glycerin intensifies the fluorescence, and the hydrochloric acid of the stomach must first be neutralized. The patient should first be given a glass of water (8 oz.—250 cc.) in which gr. 15 (1.0) of bicarbonate of soda have been dissolved. A second glass of water (8 oz.—250 cc.) is then administered, in which are dissolved the same amount of sodium bicarbonate, 1 dram (4.0) of glycerin, and gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016) of fluorescein; 1 or 2 ounces of lime-water may be substituted for the sodium bicarbonate.

As we increase the fluorescein in strength, fluorescence diminishes and colorization begins. By means of fluorescent media I have found it possible to illuminate the stomachs of fat or muscular subjects that were formerly unsatisfactory, and to examine for tumors and the location of the stomach with greater accuracy. The brilliancy of the illumination is markedly increased.

Examination of the urine of patients who have taken fluorescein shows no deleterious effects—no albumin, no sugar, no casts. The fluorescein acts in an alkaline medium and free acid destroys fluorescence, yet on catheterization of these patients greenish fluorescent urine is obtained one hour after the administration of fluorescein solution, and this condition persists for about four hours. The acidity of the urine is not due to the presence of free acid. fluorescein solution we have an additional means of testing permeability of the kidneys.

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[1] New York Med. Journal; Philadelphia Med. Journal, Feb. 13, 1904; New York Med. Journal, August 6, 1904.

The technic of gastrodiaphany is as follows: The patier stomach should be empty. He is given a glass (8 oz.—250 cc.) the alkaline solution, and then a second glass (8 oz.—250 cc.) of fluorescein solution. I frequently give *an extra half or even additional glasses of water*, especially in the suspected cases of dila tion or ptosis, or in stout subjects. In the latter, gastrodiapha is not as satisfactory, but by pressing on the abdominal wall outlines can be secured.

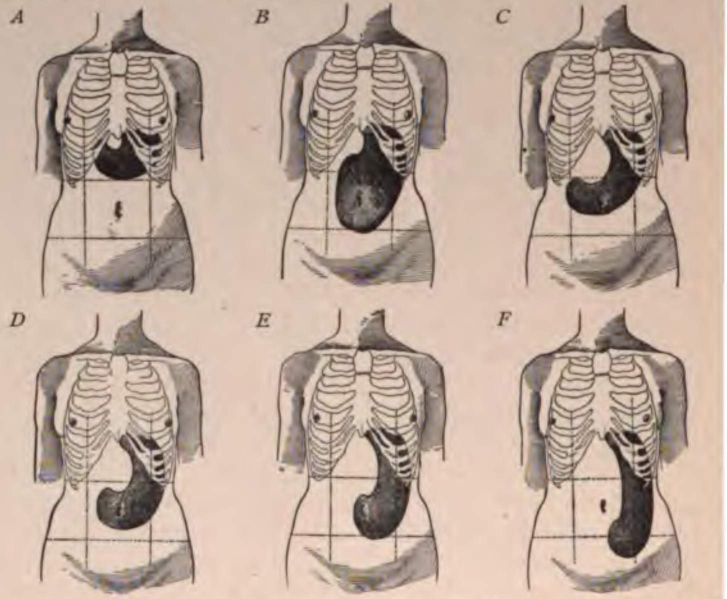

Fig. 45.—Gastrodiaphany: *A*, Normal stomach; *B*, dilated stomach; *C*, *D*, *E* varying degrees of gastroptosis (Rose and Kemp).

A dark room gives the only satisfactory results. It can devised by pinning blankets across the windows. The patient also be examined in a light room by covering him from neck to f with a dark blanket or black gown, and the examiner looking throu an opening therein.

The gastrodiaphane is introduced by gaslight or candlelig the patient sitting opposite in a chair, with the abdomen expos The electric current is turned on and the room darkened. patient should then stand up, as this position is preferable. It my custom to mark out the anatomic regions on the abdomen each case with blue pencil, and then draw the outlines of the stoma during transillumination.

With gastroptosis, the lesser curvature can be determined.

some cases the stomach will be bottle-neck above, with the base below; or somewhat pear shaped, the narrow part showing above, as the light disappears beneath the ribs. In a dilated stomach the transverse diameter of transillumination is nearly the same throughout; as we withdraw the light it begins to narrow just below the tip of the ensiform. If we illuminate in the dorsal position, the light hardly shows at all; it becomes clearer as the patient gradually sits erect, and is most marked in the standing position. This substantiates the view of Meltzing, who states that in the dorsal position only a portion of the stomach is in contact with the abdominal wall, and it demonstrates the necessity of the standing position for accurate illumination.

In Fig. 45 are shown a normal stomach, the dilated organ, and several degrees of gastroptosis. There is no question but that ptosis of the stomach exists from a very slight to an extreme degree. Tumors or thickenings of the anterior wall of the stomach, or the lesser or greater curvature, or anterior surface of the pylorus, appear as a dark area projecting into or surrounded by a light zone (Fig. 46).

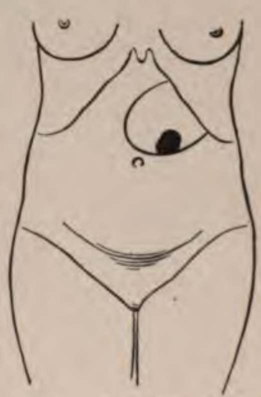

Fig. 46.—Cancer of greater curvature. Gastrodiaphany.

By the circumscribing gastrodiaphane the entire contour of the stomach is determined. The older instruments show only the lower segment clearly.

RÖNTGEN RAYS (*X*-RAYS)

The most valuable practical application of the *x*-rays[1] in reference to the stomach is for the detection of a foreign body which is believed to be lodged within the organ and for demonstrating its location. The question often of operation and the location of the incision depend upon the information secured by this means.

Cannon has made a valuable study of the motions of the stomach with the *x*-rays, to which I have alluded.

Röntgenography of the stomach has been performed by numerous observers for the purpose of locating the position of the organ.

The patient on an empty stomach is given 1 pint of milk, in which 1 ounce or more of bismuth subnitrate has been thoroughly mixed. On exposure to the *x*-rays for about fifteen seconds, from a photographic plate placed directly over the abdomen, a picture can be obtained; or with a fluoroscope the stomach can be inspected.

This method is expensive for the patient, and no information can be secured that cannot be obtained by the physical examination, especially by transillumination. The Röntgen picture shows only the greater curvature and part of the body of the stomach; the lesser curvature is not delineated. This can be demonstrated by comparing the result with subsequent gastrodiaphany.

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[1] Their use in carcinoma of the stomach and in stenosis of the esophagus are described under these topics.

By the administration of bismuth subnitrate capsules the lower border of the stomach can be delineated by the x-rays.

RADIUM TRANSILLUMINATION OF THE STOMACH

This method was first suggested by Einhorn.[1] with his radiodiaphane (Fig. 47), a rubber-covered glass capsule, containing 0.05 gm. bromid of radium (Curie, 1,000,000 strength), an inflating bulb, and Kahlbaum's fluoroscope. The transillumination is very faint and the results not as clear as from gastrodiaphany. The expense is considerable. At the present stage of its development I would not recommend it as of practical value.

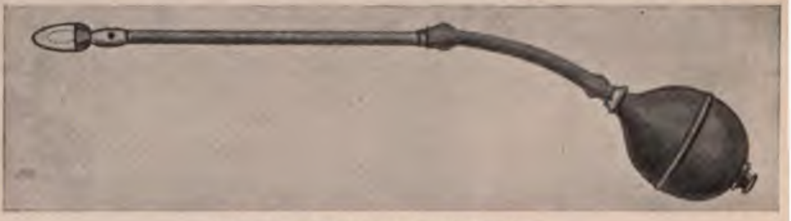

Fig. 47.—Einhorn's radiodiaphane.

RADIUM PHOTOGRAPHS OF THE STOMACH

The same may be said of this procedure devised by Einhorn.[2]

The length of exposure to radium is never less than an hour and, preferably, longer to secure results.

This is decidedly objectionable, as severe burns are possible from radium. If photographs are to be taken, the x-rays are safer and give more definite results.

CONCLUSIONS

The following methods I have found from experience to be most practical for general use:

Inspection.—By this method the peculiar shape of the abdomen, suggestive of gastroptosis, can at once be determined.

Palpation.—If movable kidney be present, it is pathognomonic of gastroptosis, especially if the lower border of the stomach lies abnormally low. Sensitive areas can also be determined and often the presence of a tumor.

Percussion.—The scratch method of auscultatory percussion is serviceable in mapping out the stomach, as is also auscultatory percussion.

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[1] Medical Record, July 30, 1904.

[2] Archives of Physiological Therapy, Sept., 1905.

Splashing Sound.—If not present, it can be artificially produced, and is most *valuable in determining the lower border of the stomach.*

Dehio's Method.—Additional water can be given if desired, and by percussion the observations determined by the splashing sound can be substantiated for accuracy.

Gastrodiaphany.—This can be employed to differentiate in doubtful cases between ptosis and dilatation, and is of value in accurately mapping out the stomach before surgical operation. By it one can at times determine the presence of a tumor at an early stage.

Inflation.—This method, especially by distention with carbonic acid gas, is an aid in mapping out the stomach and in determining the position of the upper as well as of the lower border.

CHAPTER VI

EXAMINATION OF THE FUNCTIONS OF THE STOMAC

THE determination of the functions of the stomach—secretor motor, and absorptive—is of importance for accurate diagnosi Of the examination of the secretory function I will first speak.

Ewald and Boas have demonstrated that gastric secretion begi as soon as food enters the stomach, and continues until the chyn has passed into the intestines. The secretion is diminished towa the last. Examination at various periods after taking food wi give different results, and it is necessary to examine the gastr contents at a definite time, during the height of digestion. It desirable that a definite test meal should be administered.

TEST MEALS

Riegel's Test Dinner.—Riegel's test dinner is the oldest adv cated. This consists of a plate of meat broth (about 400 cc.); beafsteak weighing from 150 to 200 gm. (5–7 ounces); 50 gm (1 ounces) of mashed potatoes; and a roll (35 gm.).

The average time one should aspirate the stomach contents i about four hours after this meal.

Ewald's Test Meal.—This consists of about 6 ounces (175 gm. of finely chopped meat; stale bread, 35 gm., and butter, to be take three hours before withdrawal of the stomach contents.

This is practical as regards quantity, as by some the test mea of Riegel is considered large in amount.

Test Meal of Germain Sée.—The patient is given 3 to 5 ounce (100–150 gm.) of white bread; 2 to 3 ounces (60–80 gm.) of finel chopped meat. and a large glass (300 cc.) of water; and the content examined two hours later.

Klemperer's Test Meal.—This consists of 1 pint (500 cc.) o milk and 2 rolls (70 gm.), given on an empty stomach. Examinatio two hours later.

Test Breakfast of Ewald and Boas.—This is given in th morning in the fasting condition, and consists of 1 to 2 rolls (35–7 gm.) and 1 cup (300–400 cc.) of tea or water. Examination on hour later.

Boas' Test Breakfast.—One ounce of rolled oats boiled i 1 pint of water, with salt to taste.

Boas advocates this, as it contains no lactic acid, and believe it should be employed when an accurate test for this acid is desired

Two shredded wheat biscuits with water (300 cc.) or a pint bowl of granose have been recommended as a convenient substitute.

The test meals that are in chief use are the Riegel test dinner and Ewald-Boas test breakfast. The latter is easily procured and can be administered during office hours—*which is the most accurate method.* It is easy to recognize therein remnants of food from the previous day. The test dinner gives better results as regards the investigation of the microscopic appearances and the study of the motor functions. *The test breakfast in many cases will be sufficient.*

It is often of service to administer a special test dinner at 7 P. M. and the test breakfast in the office at 7.30 to 8.00 A. M. It will be described under Testing the Motor Functions.

I agree with Fleiner that tea is to some an irritant. In addition, it is not of stable strength, and water is preferable. The average slice of bread from the loaf weighs 30 gm. I employ usually 2 slices (60 gm.) of bread and 250 to 300 cc. of water. One must allow for a patient of small physique and poor nutrition, for in such cases he cannot or will not take this quantity.

The *diagnosis* in most cases *should not be made from a single examination* of the gastric contents.

METHOD OF ASPIRATION OF THE GASTRIC CONTENTS

The selection of the proper type of tube is important. It should be of soft rubber, of a caliber of 30 to 32 French, to allow free exit of contents; have an opening at the tip, and one or even two lateral openings, as in Fig. 48, or the Ewald's tube (Fig. 49).

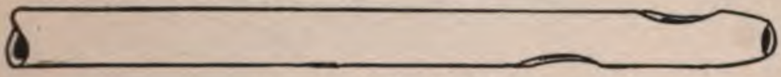

Fig. 48.—Aspirating tube.

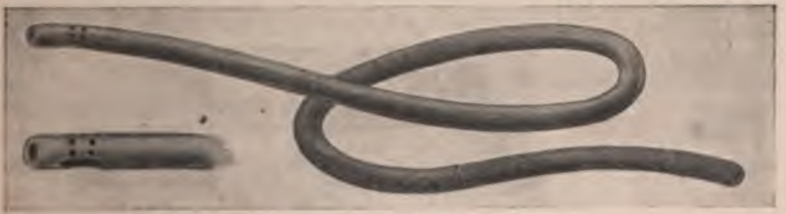

Fig. 49.—Ewald's tube.

Position of Patient and Operator.—The patient sits upright on a chair and is protected by a sheet or towel. False teeth should be removed. The operator stands in the position as depicted in Fig. 50, and passes the tube along the roof of the patient's mouth. The advantages of this method are described under Lavage.

The tube should be moistened in warm water and introduce about 20 inches until resistance is felt, when it should be slightl

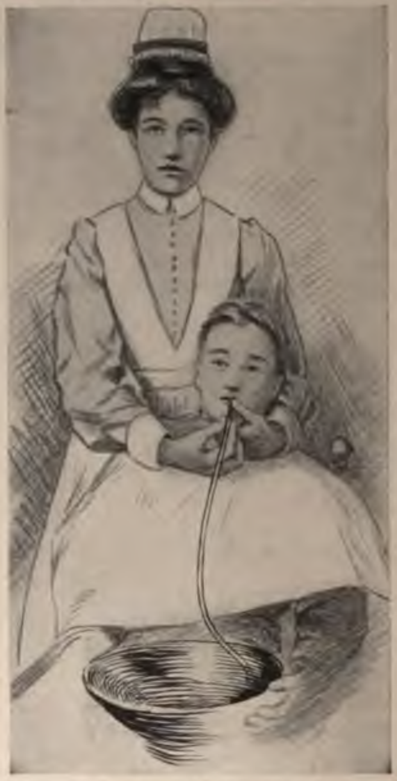

Fig. 50.—Correct position for passage of aspirating tube.

withdrawn. In the event of gastroptosis or dilatation the distance would be greater. A Politzer bulb can be employed for aspiration.

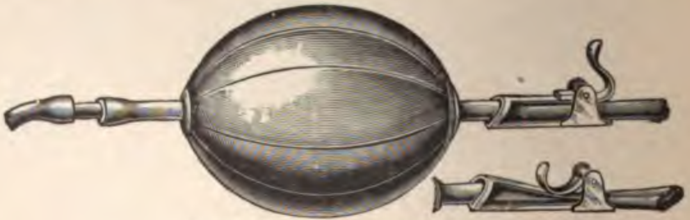

Fig. 51.—Boas' aspirating bulb.

Boas' Method.—The Boas aspirator (Fig. 51), which consists of a rubber bulb having two soft-rubber ends and provided with a clamp, is attached to the stomach-tube by a piece of glass tubing. The clamp is fixed, the bulb compressed and then released, and thus filled up; the clamp is opened, and the contents, by compression of the bulb, forced into a bottle, glass, or some other receptacle. This is a simple method of aspiration. Other devices with a vacuum bottle, mouth suction, forcing air through a double tube, etc., have been suggested, but they are more complicated.

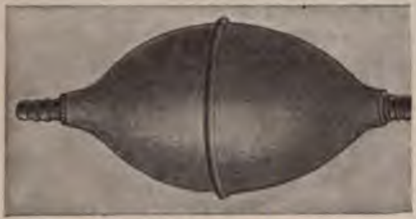

Fig. 52.—Aspirating bulb.

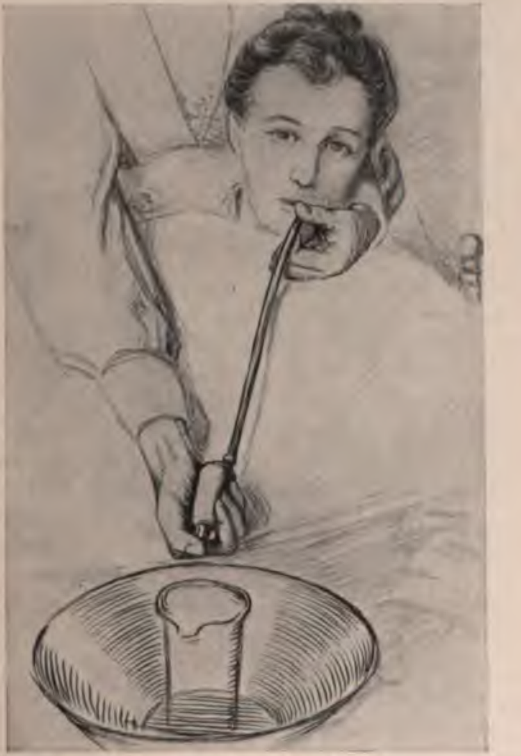

Fig. 53.—Filling the bulb.

Ewald-Boas Expression Method.—This consists in having the patient exert pressure upon his stomach by means of his abdominal muscles. He should first inspire deeply, and then compress the abdominal walls in the same manner as during defecati The pressure expels the gastric contents through the tube into a rec tacle. This method is considerably employed, and when success is unquestionably excellent. In cases of marked atony, both of stomach and abdominal walls, in excessive dilatation, and at tir from plugging of the stomach-tube, the expression method is failure.

Author's Method.—It is my custom to attach to the stoma tube a bulb without valves, such as is depicted in Fig. 52. T

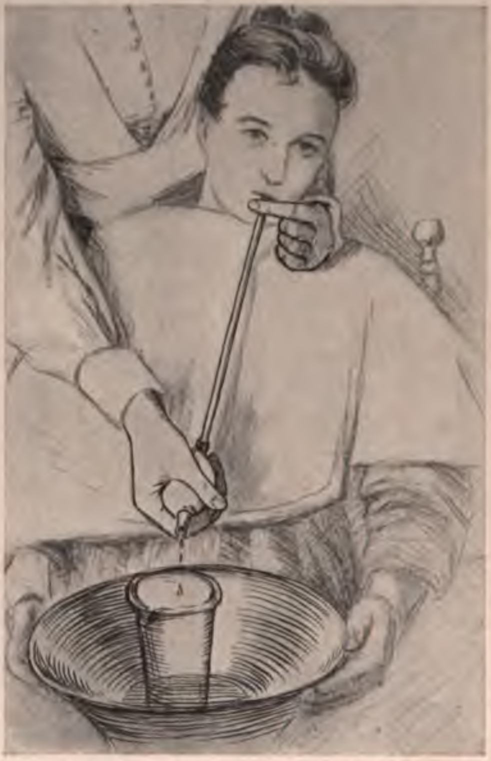

Fig. 54.—Second step of aspiration.

patient is then directed to express the contents. If the tube becor stopped, the thumb is placed over the open end, the bulb squeez and the obstruction immediately relieved. This obviates blow through or removing the tube.

If expression fails, then immediate aspiration is resorted The tube is pinched near the teeth, the bulb squeezed, and thumb placed over the open end. The stomach-tube is then relea and the vacuum in the tube allowed to fill with gastric conter as in Fig. 53.

When the bulb is filled the stomach-tube is again pinched, the thumb removed from the bulb, and the contents gradually expressed into the receiving vessel, as in Fig. 54.

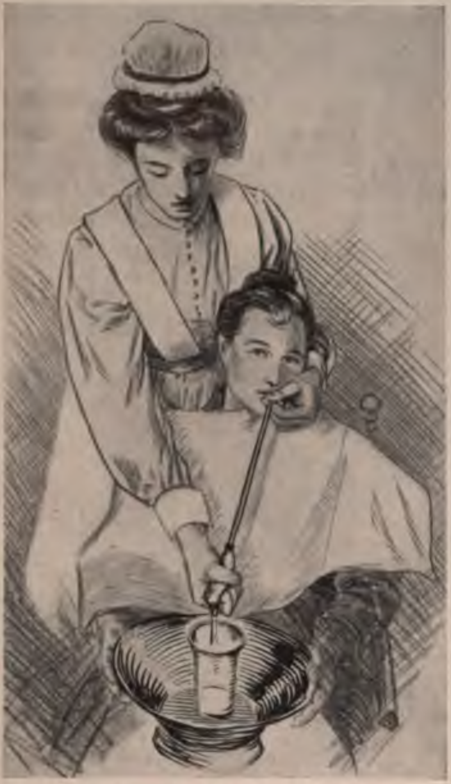

Fig. 55.—Final step of aspiration.

The final step is depicted in Fig. 55. This process is repeated until the gastric contents are removed.

EXAMINATION OF THE INGESTA

Before chemic examination of the ingesta is begun, the quantity aspirated should be carefully measured. After the Ewald-Boas test breakfast, one may expect to secure on an average 50 to 75 cc. of contents, and if 100 cc. or more be present, this would be suggestive of motor insufficiency. A large quantity of gastric contents (350 to 400 cc.) four hours after the test dinner would also be a suspicious circumstance, and further tests should be made, as described under Examination of Motor Functions. The quantity of the residue, therefore, has a chief bearing on the motor function, though in hypersecretion abnormal quantities are found.

Macroscopic inspection gives considerable information. Aft the test breakfast, in some cases large undigested pieces of bread a brought up; in others, remnants of bread that are nearly digeste or only slightly digested; and in others, a fine fluid mushy mas These findings are at once suggestive. With Riegel's test meal th differences are more pronounced; the mass may be fine, unifor and mushy, containing no coarse elements; or there may be coar undigested meat-fibers.

Mucus, blood, and bile are readily visible. In some cases th gastric contents when placed in a glass vessel forms three layer at the bottom, fine starchy material; next, cloudy fluid; and on to a foamy layer, which latter is evidence of gaseous fermentation.

Chemic Examination.—The aspirated gastric contents shoul be filtered, preferably through filter-paper. If this is not at han several layers of cheese-cloth or gauze can be employed.

From a practical point of view, the most important fact to dete mine is, whether there is an excess of hydrochloric acid during th height of digestion. If free hydrochloric acid be present, it is the necessary to find out whether the secretion is normal or increase If the hydrochloric acid is deficient in amount or absent, the digestiv power of the stomach is deficient.

When free hydrochloric acid is present, the determination o pepsin is unnecessary; in fact, it is often present, even if free hydro chloric acid is absent. In such event, if the gastric contents ar acidified with sufficient hydrochloric acid and digestion of albumi then occurs, this is evidence of sufficient pepsin formation. I cases complicated by the absence of free hydrochloric acid, th examination for pepsin should be carried out.

For a complete chemic analysis the following tests should b performed:

1. Reaction.
2. Total acidity.
3. Free hydrochloric acid.
4. Combined hydrochloric acid.
5. Lactic acid.
6. Propepton.
7. Pepton.
8. Pepsin.
9. Rennet.
10. Dextrin.
11. Erythrodextrin.
12. Achroödextrin.

Before describing the tests, it is well to remember the findings of the normal gastric juice after the test breakfast for a basis of comparison.

Normal gastric juice is of acid reaction; total acidity, 40 to 60 (0.15–0.21); free hydrochloric acid, 25 to 50 (0.1–0.2 per cent. approximately); propepton, small amount; pepton more; pepsin and rennet present; sugar and achroödextrin, present; erythrodextrin present in small amount or absent; dextrin absent.

Some patients may have free hydrochloric acid within the above normal limits, and yet suffer from the symptoms of hyperchlorhydria; while others may have free hydrochloric acid as high as 100+ and present no symptoms at all. Individual peculiarities must be considered.

Reaction.—This is determined by means of blue and red litmus-paper. If the filtrate is acid, it turns the blue paper red; and if alkaline, the red turns to blue; neutral causes no change.

Test for Free Hydrochloric Acid.—Numerous coloring-matters when exposed to the action of weak solutions of hydrochloric acid, undergo changes, and have been employed as tests for its presence. Those that are of greatest practical value I will describe shortly.

There has been considerable dispute as to the respective superiority of these tests. Though organic acids, when present in considerable quantities, may give these color changes, yet they are not as sensitive to organic acids as to mineral acids. I agree with Riegel that this danger is practically negligible. As a precaution it is well to employ one of the following qualitative check tests, which react only to free mineral acids, and not to the organic acids.

In addition, the test for lactic acid should be performed.

The Phloroglucin-vanillin Test (Günzburg's).—This reagent consists of 2 grams of phloroglucin and 1 gram of vanillin dissolved in 30 grams of absolute alcohol. An equal number of drops, 1 or 2 each, of this and the gastric juice are placed on a porcelain dish and mixed with a glass rod. The dish is then held over an alcohol lamp and the fluid allowed to evaporate slowly. A cherry-red color appears, as in Fig. 56, if free hydrochloric acid be present. If there are only traces, then there is a rose tint at the margin. If hydrochloric acid is absent, the color varies from yellow to brown.

This test responds to free hydrochloric acid and not to organic acids. The solution is *unstable*, and should be preserved in *a dark glass bottle*. It is advisable to make a fresh solution frequently.

The Resorcin-sugar Test (Boas).—Five grams of resorcin and 3 grams of cane-sugar are dissolved in 100 cc. of alcohol. Equal drops of this reagent and gastric juice are slowly evaporated to dryness, without burning, in a porcelain dish or a butter dish. If free hydrochloric acid be present, a rose-red color appears, which fades on cooling (Fig. 57). It responds to hydrochloric acid only. It is nearly as delicate, more easily obtained, less expensive, and more stable than Günzburg's test.

After performing one of these check tests it is preferable in all cases to test quantitatively for acidity; and for this purpose I prefer *Töpfer's method.*

The qualitative method gives no basis for scientific accuracy.

Töpfer's Method.—This method determines quantitativel Total acidity; free hydrochloric acid; combined hydrochloric aci total hydrochloric acid, and acid salts quite accurately for clinic purposes. In routine examinations it is rarely necessary to dete mine more than total acidity and free and combined hydrochlor acid. If free acid is absent then the pepsin and rennet tests shou be made.

Solutions Required.—(1) One per cent. alcoholic solution phenolphthalein (colorless).

(2) One per cent. aqueous solution of sodium alizarin sulphona (opaque brownish yellow).

(3) Five-tenths per cent. alcoholic solution of dimethyl-amid azobenzol (yellowish red).

(4) Decinormal solution of NaOH to titrate the gastric juic 1 cc. of tenth-normal NaOH neutralizes .00365 hydrochloric acid.

Töpfer's method depends upon the different sensitiveness of thre color end-reagents to the various constituents of the gastric juice.

Method.—Though 10 cc. of the filtrate are usually employe I have illustrated the method on the basis of 5 cc., as it is oft practically found impossible to carry out all the tests when usi larger quantities. The methods are equally correct.

In each of the three beakers (*A*, *B*, and *C*, Fig. 58) are plac 5 cc. of the filtered gastric contents.

To *beaker A* are added 1 to 2 *drops of the phenolphthalein solutic* which is used as an indicator of the *total acidity*.

This body turns red pink or red as soon as the fluid becom slightly alkaline, after the addition of the sodium hydrate.

To *beaker C* we add 1 *to 2 drops of the dimethyl-amido-azoben solution.*

A reddish-pink or cherry-red color develops if free hydrochlo acid be present, depending on the degree of acidity. After titrati with sodium hydrate, the end-reaction is a pale orange yellow.

To *beaker B* is added 1 *to 2 drops of the sodium-alizarin-sulphon solution.* After sufficient sodium hydrate is added a violet co (the end-reaction) appears.

The titration with sodium hydrate is performed from a graduat pipet or, preferably, a buret, supported on a stand. The lat should be graduated to $\frac{1}{5}$ or, by preference, to $\frac{1}{10}$ cc.

The elements combining the total acidity in beaker *A*, Fig. are free hydrochloric acid, acid salts, combined hydrochloric a and organic acids, which respond to the phenophthalein test.

In beaker *B* are free hydrochloric acid, acid salts, and orga acids, responding to the alizarin test.

Hence, as Töpfer claims, to find the *combined hydrochloric ac* one must substract the *acidity of B* from the total acidity of *A*.

In beaker *C* we have free hydrochloric acid alone, which respon to the dimethyl-amido-azobenzol test.

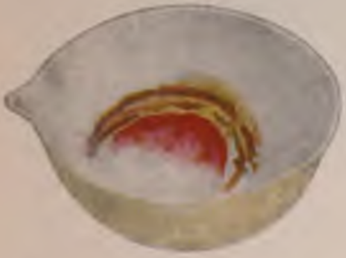

Fig. 57.—Resorcin test. Color reaction.

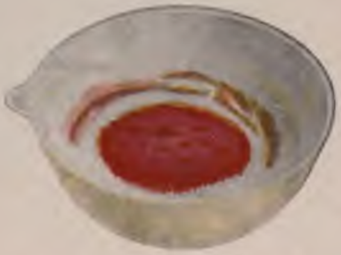

Fig. 56.—Phloroglucin-vanillin test. Color reaction.

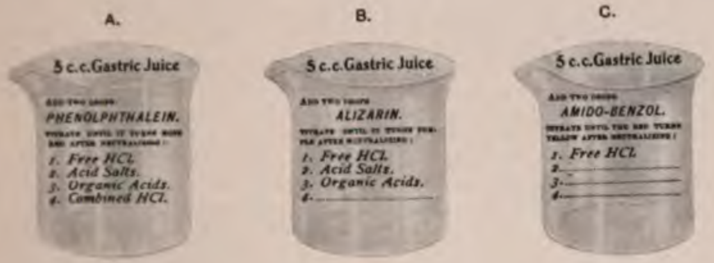

Fig. 58.

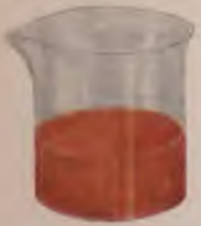

Fig. 59.—Phenolphthalein end-reaction. Total acidity.

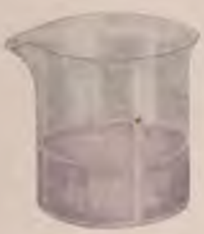

Fig. 61.—Sodium alizarin sulphonate end-reaction.

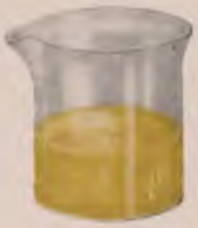

Fig. 60.—Dimethyl-amido-azobenzol end-reaction.

In the absence of organic acids, the acidity of *C* substracted from the acidity of *B* will give the acid salts.

Total Acidity.—The beakers being thus prepared, titration of beaker *A* (total acidity) is begun with the sodium hydrate solution. As this is added, the reddish color appears, but it disappears as the fluid is agitated. The procedure should be continued until a permanent red-pink or reddish color is present, as in end-reaction (Fig. 59).

As the degree of acidity is expressed by the number of cubic centimeters of a decinormal solution of sodium hydrate required to saturate or make slightly alkaline 100 cc. of the gastric contents, and as only 5 cc. of the latter was employed (which is $\frac{1}{20}$ of the total quantity), the number of centimeters of the sodium hydrate necessary to produce the end-reaction must be multiplied by 20.

Thus if 3 cc. sodium hydrate produced the end-reaction in 5 cc. of the filtrate, the total acidity would be 3×20, or 60+.

Multiply this figure of acidity by 0.00365, and we have the percentage of total hydrochloric acid, or 60×0.00365 equals 0.219 per cent.

Free Hydrochloric Acid.—Commence titration of beaker *C*, to which the dimethyl-amido-azobenzol solution has been added, and continue titration until the solution becomes a pale lemon yellow, as in Fig. 60.

As saturation with the decinormal sodium hydrate solution was computed on the basis of 100 cc. of gastric contents, and only 5 cc. or $\frac{1}{20}$ were tested, the number of cubic centimeters of sodium hydrate solution required to produce the end-reaction must be multiplied by 20.

Thus, if 2 cc. of this alkaline solution will produce this result, we must multiply it by 20, and we say the free hydrochloric acid is 40+.

To compute free hydrochloric acid in percentage, multiply $40 \times 0.00365 = 0.1460$ per cent.

Combined Hydrochloric Acid.—Commence titration of beaker *B*, to which the sodium-alizarin-sulphonate has been added, and continue the process until the end-reaction, the violet color, as in Fig. 61, occurs.

As only 5 cc. of gastric contents are employed, and again the computation is based on 100 cc., the number of cubic centimeters of decinormal sodium hydrate employed must be multiplied by 20.

If, for example, 2.2 cc. were required, $2.2 \times 20 = 44+$acidity.

Töpfer has shown that alizarin is sensitive for all the elements comprising acidity, except for the combined hydrochloric acid.

The acidity 44, therefore, secured by this reaction must be substracted from 60, the total acidity; and this gives 16 acidity as the combined hydrochloric acid.

In percentage $16 \times 0.00365 = .06$ per cent. combined hydrochloric acid.

The total acidity, free and combined hydrochloric acid, ar important to examine for as a matter of routine.

As fractions of a centimeter must often be computed, I give a example in tabulated form:

Beaker A, 5 cc. gastric contents; for ***total acidity***, 3.2 cc. deci normal sodium hydrate gives end-reaction.

Beaker B, 5 cc. gastric contents; for ***alizarin test***, 2.4 cc. deci normal sodium hydrate gives end-reaction.

Beaker C, 5 cc. gastric contents; for ***free hydrochloric acid***, 1.5 c decinormal sodium hydrate gives end-reaction.

1. *Total Acidity. Beaker A.*—$5 \text{ cc.} \times 20 = 100 \text{ cc.}$ $3.2 \text{ cc.} \times 2$ $= 64 \text{ cc.}$ $64 \times 0.00365 = .23$ per cent.

2. *Free Hydrochloric Acid. Beaker C.*—$1.5 \text{ cc.} \times 20 = 30 \text{ cc.}$ $30 \times$ $0.00365 = .11$ per cent.

3. *Combined Hydrochloric Acid = A–B.*—

$B =$ alizarin reaction. $A = 64$
$2.4 \times 20 = 48.$ $B = 48$
$A - B = 16 =$ combined HCl.
$16 \times 0.00365 = .06$ per cent.

4. *Total Hydrochloric Acid, Free and Combined.*—.11 per cen $+ .06$ per cent. $= .17$ per cent.

5. *Acid Salts = B – C (Organic Acids Absent).*—

$B = 48$
$C = 30$
$B - C = 18$
$B - C = 18$
$18 \times 0.00365 = .07$ per cent.

If 10 cc. of gastric juice be employed in the tests, then the numb of cubic centimeters of sodium hydrate required to produce the en reaction must be multiplied by 10.

If 2 cc. of gastric juice is used, the multiple is 50, and so on.

The further tests are as follows:

Lactic Acid.—*Uffelmann's Test.*—This reagent is the one mo frequently employed and is sufficiently accurate, if necessary pr cautions are observed. It should be freshly prepared before eac test. It can be prepared as follows: 10 cc. of a *4 per cent. carbol acid solution* is mixed with *20 cc. of distilled water*, and to this added 1 drop of *sesquichlorid of iron*. A watery solution of carbol acid (2 per cent.), to which is added 1 drop of liquor ferri sesqu chlorid, is another method of preparation. These solutions have a amethyst-blue color.

Other methods of preparation are recommended by Riege 20 cc. of distilled water, 10 cc. of a 4 per cent. carbolic acid solutio and 0.1 cc. of neutral 10 per cent. iron chlorid solution; or, dilut the officinal iron chlorid solution with distilled water until th solution is about colorless, and then add a 2 to 4 per cent. solutio of carbolic until the amethyst-blue color appears.

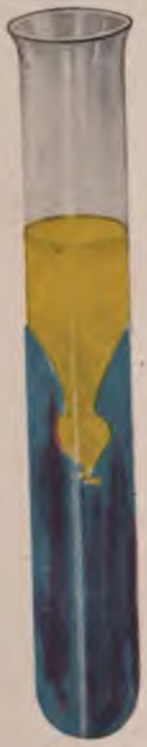

Fig. 62.

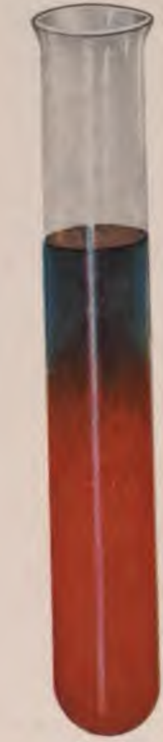

Fig. 63.

Fig. 62.—Uffelmann's test.
Fig. 63.—Congo red test.

A very dilute iron chlorid solution will also give the reaction, but the blue color acts as a contrast.

The lactic acid reaction has been described as a *canary-yellow* or, more often, a *canary-green color* (Fig. 62).

Fatty acids produce an *ash-gray* and *inorganic acids decolorize* the blue solution.

As at times the phosphates may be present in the gastric contents, and they give the same reaction, a modification has been recommended, which is *practical for general use.*

Modified Uffelmann.—Take 5 cc. of the filtrate plus 10 cc. of ether, and shake in a test-tube for a few minutes; then allow it to stand until the ethereal solution has separated from watery solution. Pour the ethereal part into another test-tube and place it in a glass of hot water to evaporate. Add 1 cc. of distilled water to the remaining drops, and test for lactic acid with the Uffelmann solution. If the canary color occurs, lactic acid is positively shown.

A larger quantity of the filtrate can be employed with two or three times the quantity of ether. Fleiner does not evaporate the ether, but adds Uffelmann's solution directly to it. The solution will appear yellow at the bottom of the tube if lactic acid be present.

Strauss employs a mixing funnel with two markings, one at 5 cc. and the second at 25 cc. (Fig. 64).

Fill to 5 cc. with the stomach filtrate. Pour on ether (Squibbs) up to 25 cc. and shake the mixture well. Open the lower stop-cock and allow the fluid to run off until it reaches 5 cc., and then pour in distilled water to 25 cc. To this mixture add 2 drops of iron chlorid solution (1 : 9 distilled water), and shake the whole.

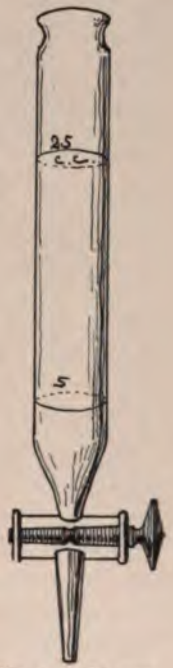

Fig. 64.—Strauss mixing funnel.

Investigations by Strauss show that if one promillimeter of lactic acid be present, an intense green color occurs; if less lactic acid, the color is light green.

Arnold[1] suggests a new test:

1. A solution of gentian violet (0.2 cc. saturated alcoholic solution in 500 cc. distilled water) and

2. Tincture ferri perchloridi, 5 cc. (U. S. P.), diluted with distilled water (20 cc.).

A drop of the iron solution gives a blue color with 1 cc. of the gentian violet, which changes to yellowish green when gastric contents which contain lactic acid are added.

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[1] Journal of American Medical Association, 1898, vol. viii, p. 21.

Other methods have been suggested, notably that of Boas, which is rather complicated. It is based on the oxidation of lactic acid into acetaldehyd and formic acid.

The presence of aldehyd is demonstrated by the iodoform reaction with alkaline iodin solution, or of aldehyd of mercury with Nessler's reagent.

Boas Method.—Take 10 to 20 cc. of the gastric filtrate and evaporate it into a syrupy consistency over the water-bath. If free hydrochloric acid is present, an excess of barium carbonate is added. A few drops of phosphoric acid are then mixed in, and the carbonic acid is expelled by boiling. The fluid is then cooled and extracted two or three times with 50 cc. of ether.

After half an hour pour off the clear ethereal layer. The ether is now evaporated and the residue washed in a flask with 45 cc. of water, well shaken and filtered. Concentrated sulphuric acid, 5 cc. (sp. gr. 1.89), and a pinch of manganese dioxid are added to the filtrate. The mixture is then distilled over a small flame, and the vapor conducted into a narrow cylinder containing 5 to 10 cc. of an alkaline iodin solution. This consists of equal parts of a decinormal iodin solution and the standard potassium hydrate solution. The vapor may be conducted into the same quantity of Nessler's reagent. If lactic acid is present it gives rise to the iodoform reaction (clouding and odor of iodoform) with the iodin mixture. If Nessler's reagent is used, yellowish-red aldehyd of mercury appears.

This procedure is further elaborated for the quantitative estimation of lactic acid, but it is extremely complicated and clinically unnecessary.

We may say that *when a test breakfast or test dinner is taken under proper conditions, only traces of lactic acid are introduced, and finding it in appreciable quantities in the gastric contents is of pathologic significance, showing subacidity and stagnation.* It is not pathognomonic of cancer, but these conditions frequently exist in such cases.

Quantitative Estimation of Lactic Acid.—The acidity of the filtrate is first determined; 10 cc. of the filtrate are shaken up with ether in excess. The ether is then separated and the degree of acidity computed therein.

Subtract this figure from the total acidity and multiply by .09, which gives the percentage.

Volatile acids must be first tested for and eliminated by boiling. This method is only approximate.

Volatile Acids.—Fatty or volatile acids are recognized by boiling a few cubic centimeters of the filtrate in a test-tube. A strip of moistened blue litmus-paper is held over the escaping vapors. The paper will turn red if they are present. Their quantitative determination is hardly necessary.

Acetic Acid.—In large quantities acetic acid can be detected by its characteristic odor. For the detection of small quantities, Einhorn neutralizes the watery residue of the ethereal extract of the gastric filtrate with carbonate of soda, and then adds neutral chlorid of iron solution, when a red color is developed.

Propepton.—Add to the gastric filtrate of, say, 5 cc. an equal quantity of a saturated solution of sodium chlorid. Propepton, if present, is precipitated. If none is formed, then add 1 or 2 drops of acetic acid, and precipitation will occur if propepton is present. On heating, the solution clears up, but again becomes turbid on cooling.

Pepton.—Preferably, after filtering out the propepton, take 2 cc. of the gastric filtrate and make strongly alkaline by adding sodium hydrate solution and then add a few drops of a weak 1 per cent. copper solution. Pepton gives a purple or violet-red color (biuret reaction).

Pepsin.—A thin disk of the white of a hard-boiled egg, weight of about 1 gram (1 cm. in diameter and 1 mm. thick), is placed in a test-tube containing 5 cc. of the gastric filtrate and kept at blood temperature. The tube can be placed in water at blood temperature in a Thermos bottle.

If free hydrochloric acid is not present in the filtrate, add 2 drops of dilute hydrochloric acid. The presence of pepsin will cause disappearance of the albumin in from two to six hours.

The methods for the quantitative determination of pepsin that have been recognized as practical for clinical purposes, are those of Hammerschlag and Mett. Henry Illoway has devised a simple method for determining the relative quantity of pepsin, which seems to the author of value: 10 cgm., exact weight, egg albumen (white of hen's egg) is coagulated in the following manner:

The egg is placed in a small pot of cold water, which is then covered with a lid and put on to boil. It is allowed to cook for ten minutes after the water has begun to boil—in all, twenty minutes from the time it has been put on. The egg is then taken out and allowed to cool, either by setting it in a saucer or by putting it into cold water.

To imitate the usual way, food is, or should be, ingested, the segment of albumin is divided into two parts. Observation has shown, as it has long been known clinically, that thus subdivided the gastric juice can act upon it more quickly. The action being from all sides, it is more effective when we have eight sides for a given quantity, than where we have only four.

The coagulated albumin is put into 10 cc. of the gastric filtrate (from stomach contents extracted one hour after an Ewald-Boas test breakfast), and this is then placed in the thermostat, which is kept at 38° C.

The time in which the 10 cgm. are digested, entirely, partially, or not at all, will give us a correct idea as to the status of the pepsin secretion in the case under examination. Illoway by experiments shows that normal digestion of the albumin requires from five to five and one-half hours.

He classifies as follows:

Hyperpepsinia.—Digestion requiring only from three to four hours, not in any pathologic sense necessarily, but only to indicate a secretion of pepsin greater than usual; which may, however, be the normal for that case.

Normal Pepsinia.—Digestion requiring from five to five and one-half hours.

Hypopepsinia.—Digestion requiring more than the usual time The degree indicated by the number of hours required beyond the standard of time.

Apepsinia.—No digestion at all.

Jacoby-Solms Method to Determine Pepsin.—*Ricin Test.*[1]—Dissolve 1.0 gram of ricin in 100 cc. of a 5 per cent. solution of sodium chlorid and filter. Mix 2 cc. of this filtrate with 0.5 cc. decinormal HCl solution; 1 cc. of diluted stomach contents is added and allowed to remain at blood temperature for three hours. Ferments clear up the ricin deposit. The quantity of pepsin is determined from the amount of dilution in which the stomach contents will cause the ricin deposit to disappear.

Solms designates the amount of gastric juice which is sufficient to clear up the 2 cc. of a 2 per cent. ricin solution in three hours, kept at the blood temperature, as one pepsin unit. The normal stomach contents contain about 100 pepsin units to the cubic centimeter.

Fuld employs a solution of edestin instead of ricin.[2]

An ordinary Thermos bottle partially filled with water at a temperature of about 100° F. can be employed in place of a thermostat; test-tubes containing the solutions being tightly corked and placed therein. Einhorn[3] employs a Thermos bottle with metal framework to hold the tubes. These last are graduated in millimeters, so as to dispense with measuring glasses.

Casein Test.—Gross[4] mixes a 1 : 1000 solution of casein containing 16 cc. of a 25 per cent. HCl to the liter with the filtrate or its dilutions, and leaves it for a quarter of an hour in the thermostat.

He then adds a few drops of the concentrated solution of sodium acetate, which results in a precipitate if the casein has not been digested; otherwise the solution remains clear.

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[1] "Ueber eine neue methode der quantitative Pepsinbestimmung und ihre Klinishe verwendung," Zeitschr. f. klin Med., Bd. 64, Heft 1 and 2.

[2] "Pepsinbestimmung vermittelst Edestins," Münchener med. Wochenschr., 1907, No. 27, Vereinsbeilage.

[3] Einhorn "A simplification of the Jacoby-Solms Ricin Method for Pepsin Determination." Med. Record, Aug. 29, 1908.

[4] "Die Wirksamkeit des Pepsins und eine einfache methode zu ihrer Bestimmung," Berliner klin. Wochenschr, 1908, No. 13, p. 643.

Mett's Method.—This consists in sucking fresh egg albumen into capillary tubes of 1 or 2 mm. diameter, coagulating the albumen by boiling, and then cutting off portions 3 to 5 cm. long of the filled tube and adding these pieces to the gastric contents. This should be kept at body temperature for ten hours in the incubator. At the end of this period each end of the tube will show a lack of solid albumen owing to digestion, while some will remain in the central portion. Both the empty portions and the portion that is full are measured, and the activity of the pepsin digestion is thus determined. The relative amount of pepsin varies according to the square of the length of the empty portion of the tube, the figures of the latter being expressed in millimeters; thus, 3 mm. of digestion equals 9 parts of pepsin; 2 mm., 4 parts of pepsin, etc.

Rennet.—Add 5 drops of the filtered gastric contents, preferably neutralized with decinormal sodium hydrate solution, to 10 cc. of fresh neutral milk in a test-tube. Place this in a glass of warm water at a temperature of about 100° F., or in a thermostat. A Thermos bottle containing warm water is extremely convenient.

Normal Rennet.—In about five to fifteen minutes coagulation will occur if the rennet be normal. If the same quantity of filtrate (5 drops) be added to 20 cc. of milk, Illoway finds, under normal conditions, coagulation will occur within fifteen to thirty minutes. He suggests a simple quantitative method.

Deficient Rennet.—Add 1 cc. of gastric filtrate to 10 cc. and 20 cc. of milk, if the smaller quantity of filtrate (5 drops) gives no result. If reaction occurs within the same period, rennet is deficient.

More marked deficiency when no result is obtained with 1 cc. of gastric filtrate, but is obtained within the half-hour with 5 cc. of filtrate.

Absence of Rennet.—When no reaction is obtained within half an hour with 5 cc. of gastric juice and 10 cc. of filtrate, no reaction will occur.

It has been demonstrated by Illoway's experiments that rennet may be present in nearly normal amount, even if pepsin is markedly deficient; it may be present even if pepsin is absent.

Rennet is usually the last one of the elements active in the process of gastric digestion to disappear.

Rennet Zymogen (Chymosinogen).—Add to the same specimen of milk 3 to 5 drops of a 1 per cent. solution of calcium chlorid and place in an incubator. Coagulation shows the presence of the enzyme; otherwise it is absent.

Examination of Starch Digestion.—The salivary ferment continues its action on starch in the stomach while the amount of hydrochloric acid is not too great. It is estimated that as soon as the total hydrochloric acid reaches 0.12, the action ceases. If the secretion of hydrochloric acid is abnormally great, starch digestion is soon stopped, and there is either no digestion of starch or the end-products are not formed. The reverse is the case in sub acidity.

To determine the intermediary stages, a dilute iodin-potassiu solution (Lugol's) is employed. It consists of iodin, 0.1; potassiu iodid, 0.2; aqua destillata, 200.0.

A few drops of the filtered gastric juice are placed on a porcela dish, and to it is added a drop or two of Lugol's solution. Th reactions are as follows:

Dextrin turns the fluid blue; erythrodextrin, a red purpl achroödextrin discolors slightly the yellow tincture of the Lugo maltose does not change the color.

For sugar or maltose, Fehling's or Trommer's test must b employed.

In normal cases, sugar and achroödextrin are present; erythr dextrin absent or present in small amount; dextrin absent.

If blue or blue-violet color appear, saccharification is deficien

Other Methods for Determining Free Hydrochloric Acid.– It seems advisable to describe a few additional practical method of determining free hydrochloric acid:

1. *Tropäolin* 00 (Merck's) in a concentrated watery solutio as recommended by Riegel. Knapp employs a supersaturate alcoholic solution of the same.

To 5 cc. of the filtered chyme add 2 drops of the tropäolin solutio Free hydrochloric acid turns it a cherry-red. Titrate with decino mal sodium hydrate until it becomes *amber*—the end-reaction.

As 5 cc. is $\frac{1}{20}$ of 100, on which the calculation is based, and it takes 3 cc. of the alkali to produce the end-reaction, free hydr chloric acid therefore $= 20 \times 3 = 60$.

Multiply 60×0.00365 to secure percentage.

2. *Mintz's Method.*—For example, to 10 cc. of the gastric filtrat decinormal sodium hydrate is gradually added, until 1 drop of th mixture no longer responds to the Günzburg reaction (phlorogluci vanillin test). A platinum loop should be employed as a dro carrier.

The amount of the sodium hydrate solution in this case shoul be multiplied by 10 to give free hydrochloric acid.

For example, if the reaction no longer appears after the additio of 3 cc. sodium hydrate, free hydrochloric acid $= 30$; percentag $= 30 \times 0.00365$, or 0.109 per cent.

3. *Boas and Moerner.*—They estimate the free hydrochloric aci by Congo paper, or by a 1 per cent. watery solution of Congo re which turns blue in the presence of this acid (see reaction, Fig. 63

Take 5 cc. of the filtrate and add 3 or 4 drops of the Congo re solution. More is unnecessary, though Boas adds 5 cc. of it.

On the other hand, Congo paper can be moistened in the filtrat A blue reaction results in each case.

Decinormal sodium hydrate is then added to the mixture, or th paper is placed in a porcelain dish and the alkali added. Titration is continued until the blue begins to turn red. The estimation is performed in the same way as before.

4. *Riegel's Method.*—Congo paper is employed in his test.

Take 10 cc. of the gastric filtrate. The Congo paper is dipped in the filtrate, giving the blue reaction of free hydrochloric acid. It is then placed on a saucer. Decinormal sodium hydrate is allowed to drop slowly from the buret into the filtrate, and a drop of the mixture is removed from time to time with the platinum loop and applied to the Congo paper. As the change in color becomes indistinct, this is controlled by dropping distilled water on the same piece of paper. The alkali is dropped on the paper until it begins to turn a violet red.

The number of cubic centimeters of decinormal sodium hydrate necessary to secure the end-reaction, say 3 cc., is multiplied by 10, giving free hydrochloric acid as 30. This is necessary, as the original figure is computed for 100 cc. of contents, and only 10 cc. were employed.

To complete the analysis and estimate the total acidity, 2 drops of phenolphthalein solution are added to the same filtrate, and titration with the sodium hydrate is continued until the color of the solution turns red (the end-reaction).

The total acidity is indicated by the total quantity of decinormal sodium hydrate used from the beginning of the first titration. For example, if in both titrations 6 cc. of sodium hydrate have been employed, the total acidity is 60, as 10 cc. of filtrate were examined.

Various modifications have been employed for these tests, which only serve to confuse the reader. Those described are the most practical.

Small booklets of Congo paper can be secured from Merck. It may be prepared by saturating filter-paper with a watery solution. It is of a reddish-pink color.

The qualitative examination for free hydrochloric acid can be made with this paper by dipping it into the filtered or unfiltered gastric contents. If free hydrochloric acid is present, it will turn blue.

Determination of Hydrochloric Acid Deficit.—Honigmann[1] and Von Noorden[2] determine the degree of hydrochloric acid insufficiency by adding a decinormal hydrochloric acid solution to the stomach contents until free hydrochloric acid can be detected by Congo paper or Günzburg's test.

Ten cc. of the filtrate are placed in a beaker and the decinormal hydrochloric acid solution allowed to flow into it gradually, the solution being well mixed. The test being continued until after repeatedly dipping the Congo paper into it it shows a bluish tinge.

The more hydrochloric acid required to secure the reaction, the less the amount of combined acid in the filtrate.

[1] Berlin. klin. Wochenschr., 1893, Nos. 15 and 16.

[2] Ibid., No. 19.

Or, about 25 drops of Günzburg's solution can be added to t filtrate, and then titration with the dilute acid follows until a r mirror appears on a porcelain dish as a couple of drops of this mi ture are evaporated over an alcohol lamp.

We know, however, that the average amount of combined hydr chloric acid is 25, or 0.1 per cent., under normal conditions, a *Töpfer's method of testing will give the required data.*

The amount of pepton and propepton qualitatively are an indic tion. When there is no biuret reaction, there will be no combin hydrochloric acid.

Other Methods of Testing the Gastric Secretion.—There a several ingenious methods for testing the gastric secretion in ord to obviate the unpleasant procedure of aspiration, or because son patients absolutely refuse the tube, or there is danger incurred fro its passage, such as in cases of aneurism, angina, severe endocardit or after a recent hemorrhage from the stomach or lungs, or in the ve debilitated.

Sahli's Desmoid Test.[1]—This consists in placing methylene-bl or iodoform in a small rubber bag and tying it tightly with thin ra catgut. The bag is swallowed after a large meal, and the urine examined for methylene-blue, or the saliva for iodin. Methylen blue colors the urine green or greenish blue. The iodin is tested f in the saliva by starch-paper and fuming nitric acid, giving a blui or violet color. It is based on the fact that raw connective tissu including catgut, is, according to Schmidt,[2] digested only by t gastric juice and not by the pancreas.

The reaction occurs in healthy persons usually six to eight hou after swallowing the bag. If it takes place later, or not at all, t secretory function is insufficient. An early reaction shows hype acidity, according to Kaliski.

Einhorn,[3] in some cases of achylia gastrica and cancer, h demonstrated that catgut is also digested in the bowel, so that t method is unsuitable.

2. *Günzburg's Method.*[4]—This is based on the same principle Sahli's. The patient swallows 0.2 gm. potassium iodid, enclosed a small rubber bag, which is tied with fibrin threads.

After the fibrin is digested, the potassium iodid is set free an absorbed. The test of the saliva is made with starch-paper and fumin nitric acid, giving a violet or bluish color. This necessitates freque examinations of the saliva, and the bag may escape and the fibr be digested in the intestine. The objections are the same as Sahli's test.

3. *Spallanzani and Edinger's Method.*[5]—They fasten a sma

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[1] Correspondenzblatt für Schweizer Aerzte, 1905, Nos. 8 and 9.

[2] Deutsch. med. Wochenschrift, 1899, No. 49.

[3] Journal of the American Medical Association, May 12, 1906.

[4] Deutsch. med. Wochenschr., 1889, No. 4.

[5] Deutsch. Arch. f. klin. Medizin, vol. xxviii, 1881.

sponge to a silk thread. The patient swallows this, and after several minutes it is withdrawn and the contents squeezed out and examined. Some of the contents are squeezed out during removal, and secretions of the esophagus and pharynx are mixed with it.

4. *Dunham's Thread Test.*[1]—The patient swallows a small tassel of test thread, colored with litmus, Congo, or dimethylamido-azobenzol, attached to a silk thread about 30 inches long. After about five minutes it is withdrawn and the color reaction noted. There are the same objections as to the previous test.

5. *Einhorn's Stomach Bucket.*[2]—This consists of a small silver capsule, open at the top, with a cross-piece as a handle, to which a silk thread is attached. Several sizes can be secured (Figs. 65 and 66).

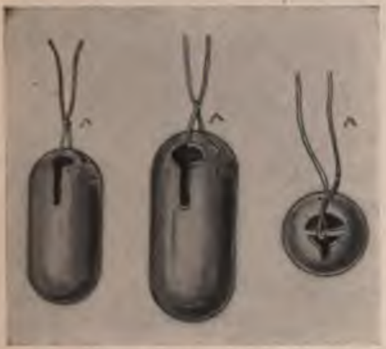

Fig. 65.—Einhorn's stomach bucket, three sizes.

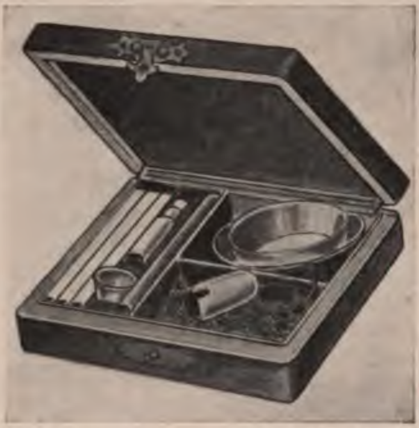

Fig. 66.—Stomach bucket set.

The bucket is moistened in warm water and emptied. It is then placed on the root of the tongue and swallowed by the patient. The vessel should remain in the stomach for about five minutes. It is then withdrawn by the silk thread.

During the act of withdrawal, if resistance is felt, the patient is told to swallow and the bucket is then readily removed.

If there are mucus and saliva in the patient's mouth, he should first gargle with plain water and expectorate the mucus.

The test breakfast should be given and the bucket administered an hour later. The gastric contents are poured from this instrument into a small beaker. Tests can then be made with blue litmus and Congo paper. A single drop can be tested with Günzburg's solution.

Einhorn demonstrates that the amount of hydrochloric acid can be approximately determined by diluting 1 drop of the filtrate with water, until Günzburg's reaction begins to disappear in the diluted fluid. He finds that normally the drop can be diluted up to eigh or ten times and give the Günzburg's reaction. The disappearanc of the reaction after five times dilution or less shows subacidity.

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[1] New York University Bulletin of the Medical Sciences, vol. i, No. 4, p. 178, Oct., 1901.

[2] Medical Record, July, 1890.

The presence of the reaction after dilution of twelve times or mo shows hyperacidity.

Recently he has employed another approximate method wit a strip of paper saturated with 0.5 per cent. dimethyl-amido-azo benzol solution and with dilution. If the paper turns red afte dilution of 1 drop of filtrate with 3 to 6 drops of water, free hydro chloric acid is normal; if no color appears after 3 drops of dilution it is subacid; and if red appears after over 6 drops dilution, it hyperacid.

If rennet is present, pepsin is also present; 2 drops of filtrat added to 2 cc. of milk placed at blood temperature in a tube in warm water will curdle in ten to twenty minutes in the presence of renne

I employ this method in exceptional cases, when the stomach-tub cannot be passed. It is preferable to aspirate.

Gas Fermentation.—The presence of free hydrochloric aci does not prevent the development of gas. It may occur in a so that is non-acid, together with lactic acid fermentation. Stagnatio is the chief factor favoring fermentation. Various gases may b formed, but from a practical point of view the determination of it occurrence, quantity, and the time necessary for its developmen are sufficient.

The unfiltered gastric contents should fill a tube such as employed for the determination of sugar in the urine (the Fiebi tube). It should be placed in an incubator at 37° C. (98.6° F. or if this is not at hand, in a uniform warm place. One can emplo a test-tube, as suggested by Moritz, closed by a rubber cork throug which is passed a bent glass tube. The test-tube is filled with gastri contents and then closed with the cork, thus forcing some of then into the curved tube and preventing the entrance of air. Th apparatus is inverted in a beaker.

As at times the sugar may already be destroyed by fermentation it is well to prepare a control tube of gastric contents to which a sma quantity of powdered dextrose has been added, or it may be adde at once. If no development of gas is noted after twenty-four hours the tube should be allowed to stand for three or four days.

Carbon dioxid may be identified by allowing a small amount o KOH to flow through a pipet to the bottom of the gas column The carbonic acid is absorbed by it and the fluid moves up to tak the place of the absorbed gas. The expressed test meal, or tes breakfast, or vomitus can be employed.

If fermentation is excessive, one can assume motor insufficiency If rapid fermentation occurs within a few hours, pyloric stenosis should be suspected, as this produces the most severe degree of moto insufficiency.

If the stomach contains a large amount of lactic acid and no free hydrochloric acid, carcinoma of the pylorus is probably the cause of this stenosis.

Gaseous fermentation is usually more intense in cases of motor insufficiency in which free hydrochloric acid is present. It can occur, however, in any form of stomach disease in which there is a disturbance of gastric secretion. Lactic acid fermentation only occurs markedly in subacid conditions. Alcohol, various hydrocarbons, and sulphuretted hydrogen have been found as products of fermentation. Boas finds H_2S chiefly in benign ectasia.

Determination of the Quantity of Chyme Within the Stomach.—Usually the quantity of chyme can be determined by emptying the patient's stomach by the expression method, especially with the addition of the aspirating bulb that I have described.

Air can be forced into the stomach by covering the free end of the bulb and squeezing the latter. If no bubbling sound is heard, the organ may be considered empty. With marked dilatation from pyloric stenosis, it is at times difficult to completely empty the organ.

Matthieu and Remond suggest the following method of determining the total quantity:

After removal of the contents, a funnel is attached to the tube and 200 cc. of water poured into the stomach. The funnel is moved up and down several times, and the patient shakes the abdomen so that complete mixture of the water and contents occurs. They are then removed by a combination of siphonage and expression.

The quantity of liquid originally contained in the stomach is equal to the number of cubic centimeters of water poured into the stomach multiplied by the degree of acidity of the second portion removed, divided by the figure resulting by deducting the degree of acidity of the second portion from that of the first plus the portion previously withdrawn.

Examination of the Vomit.—The same methods that are employed in examination after the test meal, apply to investigation of the vomitus. The information, however, is not as positive, as the admixture of bile, saliva, etc., obscures the result. Important information is secured, but the physician should inspect the vomitus in person.

If the vomiting consists chiefly of food, it should be learned when the last meal was taken and of what it consisted. If coarse morsels are found six or seven hours after a meal the conclusions are naturally different than if they were found directly after eating; and by this means we can often determine the digestive and motor powers of the patient.

If food is vomited that was ingested the day before, marked motor insufficiency is present.

In ulcer, vomiting usually occurs at the height of digestion. In some nervous diseases it usually takes place immediately after eating.

With ectasia, vomiting occurs late in digestion and often in gre quantity.

In dilatation, with hypersecretion, the fluid is abundant, an there are fine remnants of amylaceous material; while with carcinom of the pylorus undigested morsels of meat are present. In th former case free hydrochloric acid is present, and Congo paper wi turn blue; in the latter case, no color change occurs.

The presence of blood is important, and its appearance depend on the presence or absence of hydrochloric acid, on the rapidity wit which it is poured forth, and on the length of time it has remaine in the stomach. It may, therefore, look like chocolate brow coffee-grounds, or fresh blood.

Mucus is also readily discovered. Pus is rarely found, unles from perforation in phlegmonous gastritis, or from some neighborin focus into the stomach, or unless it has been swallowed.

During violent vomiting, bile is frequently in evidence, or whe vomiting occurs on an empty stomach. It may occur with pylori stenosis, when the opening is kept slightly patent. With persisten vomiting of bile one would suspect some obstruction of the duodenun such as carcinoma, torsion, gall-stones, etc.

Parasites, such as ascarides or the oxyuris vermicularis, ar occasionally found in the vomit, and very rarely a piece of gastri tumor.

Examination of the Contents of the Fasting Stomach.—I may be necessary to investigate the contents of the fasting stomach especially when disturbance of the motor function is present or whe hypersecretion is suspected. Under normal conditions one migh expect to find from 5 to even 15 cc. of gastric contents in the norma stomach. Anything over 20 cc. is considered pathologic.

The examination should be made in all cases where one suspect an abnormal quantity of gastric contents will be found in the mornin after fasting; also in all cases of suspected ectasia or atony of th stomach.

The best method is to wash the stomach thoroughly the nigh before, both in the sitting and reclining posture, and then administe a test supper. The contents are aspirated, measured, and examine the following morning before breakfast. The chief purpose is t test the motor function of the organ, especially to determine whethe motor insufficiency of a high degree is present.

When hypersecretion is suspected, the procedure is slightl different. The stomach is thoroughly washed at about 10 P. M. an care taken that all the water is removed. No food or drink ar allowed thereafter, and in the morning before breakfast the content are aspirated and examined. A quantity over 20 cc. found repeatedl I would consider pathologic (hypersecretion).

When there is permanent regurgitation of bile into the stomach, Riegel has shown that this method of examining is important. I bile and pancreatic juice enter the stomach, digestive processes are arrested.

Bile, pancreatic, and probably intestinal juice are occasionally found in the empty stomach (duodenal juice, Boas). This material is grass green or yellowish, containing bile constituents, and converts starch into maltose and dextrose, proteids into peptones, and splits fats. If it is aspirated occasionally, it is probably not significant; but if there is constant regurgitation, it is suggestive of obstruction in the duodenum.

When there is obstruction to the flow of the intestinal contents, or a communication between the stomach and intestine, intestinal contents are found in the stomach.

ABNORMAL CONSTITUENTS OF THE STOMACH CONTENTS

Abnormal products which are of importance for our diagnosis are quite frequently found in the gastric contents. They may contain mucus, blood, bile, intestinal juice, and pus.

Mucus when present in considerable quantity is easily recognized. It generally occupies the upper part of the fluid, appearing in swollen glassy lumps or in flakes and shreds. It is also intimately mixed with the food. It can be readily lifted with a glass rod. If in small amount, a few drops of dilute acetic acid are added and it will be revealed by the characteristic precipitate.

Bile and Intestinal Juice.—Small quantities of bile and intestinal juice may occasionally be met with normally. In the paragraph on "Examination of the Contents of the Stomach after Fasting" I referred to the presence of bile and intestinal juice and that their frequent occurrence is due either to relaxation of the pylorus or stenosis of the duodenum below the mouth of the bile-duct. Pure bile is golden yellow, but green if mixed with gastric juice. I believe that too often the diagnosis is made by simple inspection. Mould may produce a greenish color, and I have found it on several occasions. For accuracy, the tests should be made; Gmelin's for bile-pigment and Pettenkofer's for bile-acids.

Einhorn suggests the following for the intestinal juice, which is recognized by its ferments, trypsin, amylopsin, and steapsin:

Trypsin.—Mix the filtrate with 1 per cent. solution of sodium carbonate until the reaction is decidedly alkaline; add a flake of fibrin and keep in a warm place for several hours. Trypsin will dissolve the fibrin.

Amylopsin.—Starch is changed into maltose.

Steapsin.—Add 1 drop of blue litmus tincture and a few cubic centimeters of the neutralized filtrate to a small portion of milk and keep at blood temperature. Steapsin changes the blue color and the milk becomes slightly reddish from decomposition of the fat into fatty acids.

Blood.—Blood in large quantities is easily recognized, as is fresh blood, even if in small amounts.

Fresh blood mixed with gastric contents presents a reddis appearance, while old blood is brownish or of a coffee-ground colo It may even appear blackish. When the blood cannot be detecte microscopically and gastric hemorrhage is suspected, *occult bloo* must be examined for.

It is advisable to make the same *examination of the stool.*

1. **Benzidin Blood Test for Gastric Contents and Stool.**—Th is the most recent test devised by O. and R. Adler.[1] They fir applied it to test the feces. Schlesinger and Holst[2] advise boilin the gastric filtrate and testing in the same manner as for feces.

Gastric Contents.—Solution 1.—Knifepointful of benzidin (Merck' is added to 2 cc. of glacial acetic acid and allowed to stand an dissolve.

Solution 2.—Ten to 12 drops of the benzidin solution are adde to 2½ or 3 cc. of a 3 per cent. peroxid of hydrogen solution.

Three or 4 drops of the *gastric filtrate,* which has been boiled f about half a minute, are added to Solution 2. In the presence blood, a green or blue color results in from a few seconds to a minut

Stool.—The stool should also be examined. A small piece of fec about the size of a pea is mixed with 2 cc. of water and boiled in test-tube closed with cotton for half a minute; 3 or 4 drops of t boiled fecal solution are added to Solution 2. A green or blue col results if blood be present.

Benzidin Test Paper.—Einhorn[3] has devised a benzidin testi paper as follows:

Take a saturated solution of benzidin and glacial acetic aci moisten filter-paper therein and dry it. Both in preparing the pap and making the test avoid contact with the fingers, as a drop perspiration causes the reaction. When handling the paper empl ivory-tipped forceps or protect the hand by a towel.

Method.—Gastric Contents.—A piece of benzidin paper is fir immersed in the gastric filtrate, and then a few drops of hydrog peroxid are added. The paper is then placed on a piece of whi porcelain and examined for the development of a blue color. blood is present, a blue or green color occurs in *a few seconds to minute.* Einhorn shows that if we wait longer periods, other su stances may cause the reaction, also in time the paper moisten with peroxid will become blue.

Feces.—In testing for occult feces with the paper, a small pie the size of a pea is rubbed up with 2 cc. of water, the benzidin pap immersed therein, a drop of hydrogen peroxid added, and the bl color then examined for.

The benzidin paper Einhorn recommends as a preliminary, a if there is immediately a strong reaction or none at all, he regards t result as reliable. If at the end of a minute only a trace of reaction occurs, then the aloin-ether extract method may be employed as a check.

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[1] Zeitschr. für physiol. Chemie, Bd. 41, Heft 1 and 2, p. 59.

[2] Deutsch. med Wochenschr., 1906, No. 36, p. 1444.

[3] "A New Blood Test," Med. Record, June 8, 1907.

In the fecal examination, ether extract of feces, as employed in the aloin test, makes the benzidin test more reliable.

In *examination of the gastric contents* no meat products or iron preparations should be taken *for at least twenty-four hours before the test*, and in testing the stool *the same rule must be osberved*; but, preferably, *for a longer period if possible*, at least two to three days as a precautionary measure.

2. **Weber's Modification of Van Deen's Test for Occult Blood.**—Dilute the *stomach contents* or, preferably, the *filtrate thereof* with ⅓ volume of glacial acetic acid, and extract with about 10 cc. of ether. A few cubic centimeters of this acid ether extract are mixed with 10 drops of tincture of guaiac and 20 to 30 drops of ozonized oil of turpentine (old turpentine exposed to air).

If blood is present, the mixture turns a blue or blue violet; if absent, it turns a reddish brown with a green tinge. The reaction is more distinct if a little water is added and the blue pigment extracted with chloroform.

The Stool.—The test for occult blood is as follows:

Treat 5 cc. of feces with 20 cc. of ether; the latter is then poured off; 2 cc. of glacial acetic acid are added to the feces and thoroughly stirred. This mixture is again treated with about 10 cc. of ether and allowed to separate.

To 2 cc. of the etherized extract add 2 to 3 drops of a fresh tincture of guaiac. Then add 20 to 30 drops of ozonized oil of turpentine, or pure hydrogen peroxid, and shake well. If blood be present, there appears a blue or blue-violet color.

Meat and iron preparations should be avoided for from twenty-four to seventy-two hours before these tests.

3. **Klunge's Aloin Test.**—*Feces.*—In this test freshly prepared aloin is employed. Dissolve as much aloin as can be placed on the tip of a knife blade in 10 cc. of 70 per cent. alcohol; add 2 cc. of the aloin solution to 2 cc. of the ethereal extract of feces, prepared as above, and then the oil of turpentine or peroxid of hydrogen, as described. A cherry-red color appears in the fluid if blood be present.

Gastric Contents.—Ethereal extract of the filtrate is prepared as in Weber's test. Rest of test is same as aloin test of feces.

Einhorn at times employs aloin paper prepared with filter-paper saturated with a solution of aloin in 70 per cent. alcohol, the paper being then dried for future use. I prefer the benzidin or Weber's test.

The spectroscopic test, hemin test, etc., have been suggested, but the ones described are the most practical.

Pus is seldom found in the gastric contents and is recognized readily under the microscope. Excluding ingested pus and phlegmonous gastritis, pus shows ulceration of the gastric mucosa.

MICROSCOPIC EXAMINATION OF THE GASTRIC CONTENTS

The relative value of the microscopic examination of the gastric contents after the test meal, of the vomitus, and of the fasting stomach contents, as compared with gastric analysis, is still a matter of dispute.

Some of the ardent advocates of the microscope go so far as to claim that their method is alone necessary. Undoubtedly, in many cases the clinical symptoms, gastric analysis, the test of the motor function, and macroscopic inspection of the contents afford sufficient information for diagnosis. I do not wish, however, to depreciate the value of the microscope, as in some cases it is of fundamental importance.

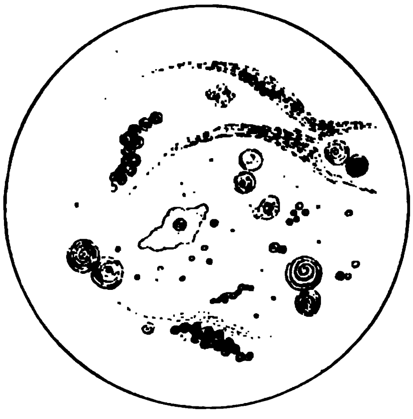

Fig. 67.—Fasting gastric juice containing mucus, snail forms, epithelial cells, and amorphous material.

Gastric Secretion.—When fasting, the gastric secretion shows normally under the microscope epithelial cells, cell nuclei, some mucus, amorphous material, and micro-organisms. Jaworski[1] describes spiral or snail-like bodies in cases of hyperchlorhydria, but Boas believes they are quite common and that they are developed from the mucus by the action of the gastric juice (Fig. 67). Einhorn has found them in patients with normal secretion.

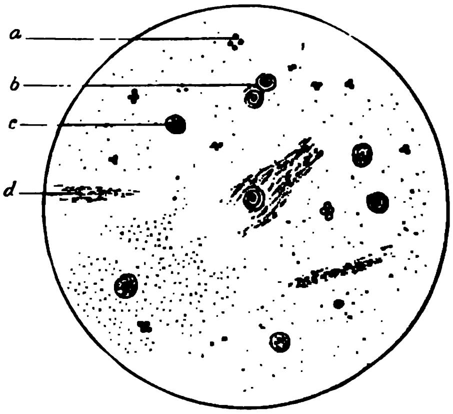

Fig. 68.—*a*, Nuclei of leukocytes; *b*, spiral bodies; *c*, nuclei of epithelial cells; *d*, striated mucus.

Mucus.—Mucus from the bronchi and lungs is characterized by the presence of alveolar cells and myelin drops; while the occurrence of a great many columnar epithelial cells is evidence of the origin from the gastric mucous membrane.

In *doubtful cases* the microscope will thus determine the origin of the mucus, either by examination of the fasting contents or after a test meal. The clinical symptoms and macroscopic appearance of the contents, as described under chronic gastritis, will, however, generally give sufficient information.

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[1] Münch. med. Wochenschr., 1887, No. 30.

Paul Cohnheim holds that the presence of free nuclei of leukocytes and epithelial cells is a positive evidence of hydrochloric acid and pepsin (Fig. 68).

I believe that gastric analysis is much preferable for such determination. Importance has been attached to the presence of two varieties of infusoria, the Trichomonas hominis and Megastoma entericum, notably by Cohnheim (Fig. 69). He believes that *their presence is pathognomonic of carcinoma*, not *affecting the motility of the stomach.* Amebæ are often associated with them.

The development of these infusoria requires the absence of hydrochloric acid, an alkaline medium, and the existence of deep folds in the mucosa. Previous to aspiration, the stomach-tube and receptacle should be warmed.

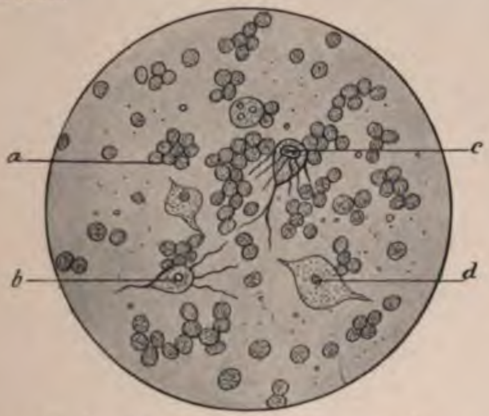

Fig. 69.—*a*, Pus cells; *b*, trichomonas; *c*, megastoma; *d*, pavement epithelium.

To differentiate between cancer and achylia gastrica, in the suspected cases, with emaciation, gastric distress, and achylia, special examination for these infusoria is advocated by Cohnheim. The presence of pus,[1] especially if associated with blood in the non-fetid gastric contents, he also believes aids in the early diagnosis before the tumor is palpable. Phlegmonous gastritis and the ingestion of pus from above must be excluded.

Sarcinæ and the Boas-Oppler bacillus can be examined for, both in the fasting stomach and after the test meal. The same is true as regards epithelial cells and pieces of the gastric mucosa. I will refer to them shortly.

Gastric Contents.—The microscopic examination of the gastric contents after the test breakfast or dinner shows, under normal conditions, a few starch granules, many of which no longer appear in spiral form. The muscular fibers do not show their diagona stripes; globules of fat, plant-cells, and micro-organisms are presen in small numbers.

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[1] Microscopic pus in the gastric contents shows ulceration, not necessarily malignant in type.

Many unchanged starch granules are found in hyperchlorhydri and hypersecretion, and the muscle-fibers are well digested; whil with hypochlorhydria (deficient secretion) unchanged muscle-fiber are present. The granules of starch are brought out clearly by th addition of one drop of tincture of iodin, giving a blue reaction. Th microscopic findings are here confirmatory of macroscopic inspectio

The varieties of micro-organisms have been thoroughly studie by DeBary[1], Nencki,[2] Boas,[3] and others. It has been demonstrate that they may be present even in hyperchlorhydria, showing tha the hydrochloric acid does not always prevent fermentation.

J. Kaufmann[4] has described a case of hyperchlorhydria in whic the motor function of the stomach was not markedly disturbed, bu which showed fermentative processes. He isolated eight varietie of micro-organisms in a specimen of the gastric contents.

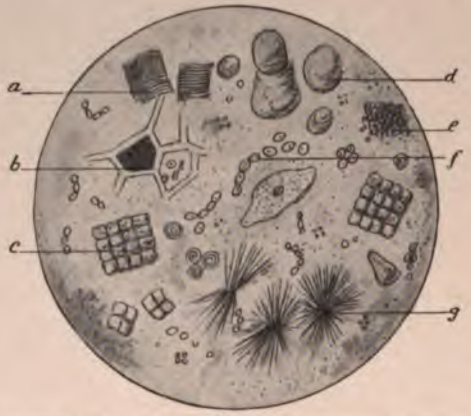

Fig. 70.—Benign ectasia. Yeast-cells and sarcinæ are prominent: *a*, Muscle fiber; *b*, plant-cells; *c*, sarcinæ; *d*, starch granules; *e*, degenerated sarcinæ; yeast-cells; *g*, fat crystals.

Boas has also described the development of sulphuretted hydroge in a case of hyperchlorhydria. In general, we may say that *fermentation develops in cases when the motility of the stomach is reduced*

Minkowski[5] has shown that if free hydrochloric acid be abundant yeast and thread fungi may be found; while if it is absent, numerou *mould organisms* are present. This last corresponds to the finding of A. Rose and myself.

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[1] Arch. f. Exper. Path. und Therap., Bd. 20, p. 243.

[2] Archiv. f. Exper. Patholog., Bd. 28.

[3] Deutsch. med. Wochenschr., 1892.

[4] Berlin. klin. Wochenschr., 1895, No. 6.

[5] See Naunyn, Mittheilungen aus der medicin. Klinik zu Königsberg, Leipsic, 1888.

Yeast.—A few isolated yeast-cells are found in the normal stomach. In ectasia or atony of marked degree the yeast-cells are numerous, arranged in colonies, and are in active process of germination (Fig. 70).

Sarcinæ.—These occur usually in cubes or bales, and are only pathologic if present in large numbers, as in benign ectasia or atony (Fig. 70), in the presence of hydrochloric acid.

They are *rare in ectasia from cancer*, and, if present, occur usually in cancer developed on an ulcer. Their presence in large numbers is an aid to diagnosis.

Boas-Oppler Bacilli.—They are unusually long, non-motile bacteria, and are characterized by their large size and end-to-end arrangement (Fig. 71).

They must be differentiated from the Leptothrix buccalis, by Gram's solution, with which they stain brown, the Leptothrix, blue. Kaufmann's[1] investigations prove the Boas-Oppler bacillus has the power of generating lactic acid from different sugars. Stagnation with lactic acid fermentation is *not specific* of pyloric carcinoma, but depends on the absence of hydrochloric acid and the presence of stagnation. These conditions exist in stenotic gastritis.

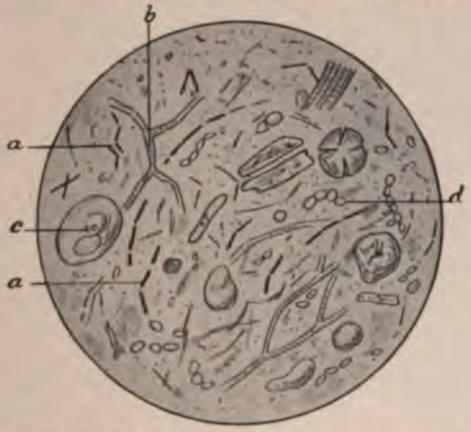

Fig. 71.—*a*, Boas-Oppler bacilli; *b*, leptothrix; *c*, potato-cell; *d*, yeast-cells.

These bacilli have been found occasionally in stomach contents that contain free hydrochloric acid (Rosenheim). Kaufmann has demonstrated them in 19 out of 20 cases of carcinoma.

The presence of these bacilli, with pronounced lactic acid fermentation and taken in connection with clinical symptoms, is very significant.

Epithelial Cells, Particles of Tumors, and Fragments of Gastric Mucosa.—Single cells cannot be diagnosed as cancer cells, but cell-nests must be found. These are rare.

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[1] Wiener klin. Wochenschr., 1895, No. 8.

Occasionally after aspiration of the fasting stomach, or test meal, or after lavage, or in the vomitus, small particles of tissue may be found, which on staining may reveal the nature of a tumor.

Hemmeter recommends in suspected cases, first, thorough lavage and rectal feeding for a day; then, passing the tube, moving it about actively, and subsequent aspiration of the fasting stomach. This is to be followed by lavage. All tissue fragments are to be examined.

Einhorn finds, especially after lavage, or at times after aspiration, small pieces of mucous membrane which he stains and examines. Some point to erosions, others to other affections. The fragment may be normal mucosa, or there may be proliferation of the connective tissue, or of the glands, or atrophy (partial or complete), or vacuolization. These conditions are illustrated in their appropriate chapters.

A positive judgment cannot be given from this examination, as only a small area may be actually involved and no changes observed in the gastric secretion. On the other hand, a bit of normal mucosa may be aspirated from a diseased organ.

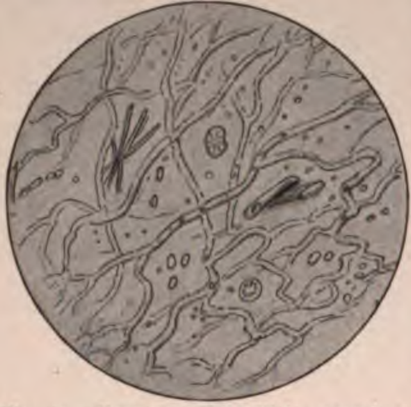

Fig. 72.—Green mould follicle, mycelia, spores, and crystals.

Mould. — Mould in the stomach has been little referred to in literature as a pathologic condition except by Talma, A. Rose, Naunyn, Einhorn,[1] and Knapp.[2] In an article on "Dilatation of the Stomach"[3] I have already referred to it.

Einhorn has found it in the wash-water of the empty stomach, as blackish-gray or brownish-green flakes of varying number; while Knapp describes it as coloring the chyme a yellowish green or dark red, and states that it has been mistaken respectively for bile or blood from its macroscopic appearance. He emphasizes the necessity for appropriate tests for bile and blood, and not the diagnosis from appearances. I can substantiate this in one case at least, in which the reddish-brown material proved to be mould.

The mould flocculi consist of clusters of spores and mycelia, sometimes mixed with mucus and epithelial cells. Crystals are also found, which Knapp considers to be segments of mould filaments.

The mould generally found has been identified by E. K. Dunham as the penicillium glaucum, though Knapp reports in addition the oïdium albicans and the aspergillus groups. These fungi probably adhere quite closely to the mucosa and may involve considerable areas (Fig. 72).

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[1] Medical Record, June 6, 1900.

[2] Organacidia Gastrica, Sept. 6, 1902.

[3] Medical News, Aug. 16, 1904.

Einhorn reports mould formation in hyperchlorhydria, in some cases attended with hypersecretion, and also in gastralgia with normal or reduced gastric secretion.

Knapp holds that the presence of organic acids in the stomach have a decided bearing, and that succinic acid and mould go hand in hand, the presence of the former being conclusive. His test for succinic acid is as follows:

Extract 1 cc. of filtered chyme, with 4 cc. of ether, and float this extract on a solution of ferric chlorid (1 drop of a 10 per cent. ferric chlorid to 2 cc. of distilled water) in a narrow test-tube. At the line of junction is a dark mahogany-red ring. He further describes symptoms in many respects resembling severe hyperchlorhydria with spasm of the pylorus, and believes the condition influenced by saccharine material in the chyme.

In the experience of A. Rose and the author, *diminished motility of the stomach* is a marked factor in favoring the growth of mould. I have found it in cases of atonic ectasia, producing many of the symptoms of chronic gastritis, and again in benign stenosis with hypersecretion. The subject requires still further investigation.

Rose finds the administration of ♏j (0.059 cc.) doses of beechwood creosote or carbonate of creosote gr. v (0.3) t. i. d. of value, and lavage is, of course, indicated, preferably with warm water—1 liter, followed by lavage with nitrate of silver 1 : 2000, or spraying the stomach with the latter, as suggested by Einhorn.

The general treatment should be according to the other conditions present.

DETERMINATION OF THE ABSORPTIVE FUNCTION OF THE STOMACH

The absorptive function of the stomach is usually tested by the method of Penzoldt and Faber, as follows: 0.2 of potassium iodid is administered in a gelatin capsule, and the saliva or urine examined every minute or two with starch-paper and fuming nitric acid. The strip of paper is moistened with saliva or urine and then touched with a drop of the acid. A *violet or blue color is the reaction.* It takes six and one-half to fifteen minutes before the reaction appears in normal conditions. In pathologic cases it is retarded.

The test should be made on an empty stomach, as with the full organ it is retarded. It is self-evident that the gastric digestion of proteids—and hence their absorption—is delayed in cases of subacidity or anacidity, while these conditions are not so important for the digestion of carbohydrates.

In some cases the *absorption of iodid is normal*, though we *know proteid digestion* is interfered with, so that I do not consider the test in every case reliable. Hershell gives a capsule containing 2 decigrams of powdered rhubarb. Under normal conditions the urine gives a red color with liquor potassæ. They are, however, the best tests so far known.

Motor Functions of the Stomach.—By this we mean the *peristaltic action of the stomach* which *expels its contents* into the intestine. The impairment of the motor power is *fully* as and in many cases *more important* than damage to the secretory functions

The best method of testing the motor power is by the test meal Leube's is the oldest method. He administered a plate of soup, a beefsteak, and a roll. If the stomach was found empty seven hours later and nothing could be washed out, it indicated that its motor power is sufficiently active. If the food remains in the stomach longer, the motor power is reduced.

We must remember that in pyloric stenosis the motor power may be really increased in endeavoring to overcome the obstacle but that food remains in the stomach for an abnormal length of time strictly speaking, it is a relative motor insufficiency. If five hours after a test meal, a small amount of chyme is aspirated, the motor power is good. If large quantities are found six hours after the meal the motor function is absolutely (or if stenosis, relatively) decreased. Greater degrees of insufficiency may be present. For example, lavage is performed and a test supper administered, say at 12 P. M. and the contents aspirated seven or eight hours later In one case there may be a small quantity of food remaining, and in another case a large amount; while in another, none. I have found chopped spinach, a few raisins, or a piece of fig an addition to the test, as they are readily recognized. Boiled rice can be added.

Boas recommends cold meat with rolls and butter and a large cup of tea.

It may be more convenient to follow the same procedure, but give the meal at lunch time. Some employ the *test breakfast. Two hours later* the stomach *should be empty.* If 100 cc. or more are found at the end of an hour, or varying quantities at the end of two hours, it shows different degrees of motor insufficiency. The test meal is more accurate. I sometimes administer a test supper and aspirate in the morning to test the motor function, following immediately with the test breakfast to examine the secretory function.

Salol Test (Ewald and Siever's Method).—Salol is not decomposed in the stomach, but in the alkaline medium of the intestine. Here it is split up, and the salicylic acid is absorbed and eliminated in the urine as salicyluric acid. The latter is recognized by testing the urine with neutral ferric chlorid solution, which gives a violet color with this acid.

The patient takes salol 1.0 (gr. 15) in two gelatin capsules half an hour after a light meal. The bladder is first emptied. Thereafter he urinates every half hour for about two hours, and the different specimens of urine are tested with the iron solution. Under normal conditions the reaction appears in from thirty to seventy-five minutes. In retarded motility, it takes two hours or more.

Ewald treats the urine with ether and examines the ethereal residue; while Einhorn moistens a piece of filter-paper with the urine and touches the middle of it with the iron solution.

Huber suggests to determine the time required for the complete disappearance of the reaction in the urine. The longer the time required for the salol to be absorbed and entirely eliminated through the urine, the longer it has remained within the stomach. He found that normally the excretion of the salicyluric acid after salol was administered lasted twenty-four hours; in patients with diminished motor function it lasted forty-eight hours or more.

Iodipin Test.—Iodipin is decomposed in the intestine. Heichelheim gives 1.6 gm. iodipin in gelatin capsules at breakfast. The saliva is then examined every fifteen minutes for iodin by starch-paper and fuming nitric acid. In normal cases the reaction appears within an hour.

Klemperer's Oil Test.—Oil is not absorbed in the stomach. After washing the stomach, 100 cc. of pure olive oil are poured into the empty organ. Two hours later the stomach is thoroughly aspirated. The difference between the original quantity of oil and that withdrawn indicates the condition of the motor function. Normally at this time only 20 to 40 cc. of oil should be aspirated.

Einhorn's Gastrograph.—Einhorn has invented a deglutible ball, arranged with an electric circuit, so that the movements which mix and break up the food can be registered.

Hemmeter-Moritz Method.—This method has been independently employed by Hemmeter and Moritz.

A thin deglutible bag is attached to an esophageal tube. The bag is then blown up and connected with a tambour on the Ludwig kymograph, which registers all the movements with a pen. A pneumograph is tied about the chest to record the respiratory movements as a basis of comparison. The muscular contractions of the stomach are demonstrated on the record as independent of the respiration.

These instruments are of interest scientifically, but their practical value has not been demonstrated.

CHAPTER VII

DIET

THE study of nutrition, both in health and disease, is importar but it will only be possible to enunciate the general principles.

There are three groups of food stuffs—proteids, carbohydrat and fats—procured from the animal and vegetable kingdoms, whi in combination furnish the most suitable form of nourishmer Climatic conditions and environment have an influence on t requirements for nutrition. In extremely cold climates the Esq maux have lived for many generations on nearly an exclusive nitrogenous diet, in which fat, which produces the greatest numb of heat units, predominates. In hot regions many of the races li principally on a non-nitrogenous diet. We also know that veg tarians live and thrive on carbohydrates.

A mixed diet is the most suitable form of nourishment.

DIET IN HEALTH

Voit has emphasized the fact that the smallest amount of protei with non-nitrogenous food added, that will keep the body in co tinual vigor is the ideal diet. He holds that a healthy adult of ave age weight should ingest 100 gm. of albumin, 50 gm. of fat, ar 450 gm. of carbohydrate in twenty-four hours; others place t requirement for proteid as considerably higher. A small proporti of the food serves the purpose of reconstructing the tissue wast while the major part is used for generating the heat required for t maintenance of life. It is, therefore, customary to speak of t necessary amount of heat units during the twenty-four hours instea of the quantity of food.

A calorie (or heat unit) may be defined as the amount of he required to raise 1 gram of water 1° C. This is a small calori A large calorie is the amount necessary to raise 1 kilogram of wat 1° C. Hence a large calorie equals 1000 small calories.

1 gm. carbohydrate yields 4.1 large calories.
1 gm. fat " 9.3 " "
1 gm. proteid " 4.1 " "

In order to calculate the calorie value of any kind of food, the num ber of grams of albumin that are contained in it are multiplied b 4.1; the grams of carbohydrate by 4.1; the grams of fat by 9.3. Thes are added together and give the total calorie value of the food For example:

100 gm. albumin × 4.1 = 410 calories.
50 " fat × 9.3 = 465 "
450 " carbohydrate × 4.1 = 1845 "
2720 total calories.

The calorie value of vegetable proteid is slightly less than that of animal proteid; 50 gm. of fat about equal 113 gm. of starch in calorie value.

Riegel holds that a human being at rest demands about 35 calories per kilogram of his body weight, and a person performing light work about 40 calories per kilo. From this estimate, the calorie value of the food of an individual weighing 50 kilos is from 1750 to 2000 calories. The weight of the patient must, therefore, be known in order to select the correct amount of nourishment.

Rubner states that different articles of food can replace each other according to their calorie value, and that it is immaterial in what form the calories are introduced into the organism. This may be taken advantage of temporarily in certain pathologic conditions when it is necessary to limit some special variety of food, such as the carbohydrates. On the other hand, a certain amount of proteid is necessary for the organism, while *unquestionable damage* can be done *by excess* in this direction.

The results of scientific study are opposed to the prevailing dietary standards, especially in regard to proteid foods. It is true that no other form of food can take the place of proteids, for a certain quantity is needed each day to replace the loss of tissue material broken down, and our choice of the varied articles of diet should be regulated by the amount of proteid they contain. It is not necessary, however, that they should exceed the other foods in amount or approach them in quantity.

Russell H. Chittenden[1] has clearly demonstrated by his scientific researches that the recommended dietary standards are excessive in quantity,[2] especially in regard to proteids. They do not undergo complete oxidation in the body like non-nitrogenous foods, but there is left behind a residue of non-combustible matter, crystalline nitrogenous products, which ultimately, if occurring in excess of the requirements of the body, prove injurious to the gastro-intestinal tract, liver, kidneys, and nervous system.

The fats and carbohydrates are easily eliminated, becoming carbonic acid gas and water. Overfeeding with a tendency to obesity is an evil, as the fat acts as a mechanic obstacle to the activity of the body and interferes with the movements of the heart and other organs; and, in addition, fatty degeneration may occur. *Potential energy*, however, can be fully as advantageously met by the non-nitrogenous foods, carbohydrates, and fats.

Fletcher has demonstrated on himself that deliberation in eating, necessitated by the habit of thorough insalivation, results in the occurrence of satiety on the ingestion of a comparatively small amount of food, and hence excess is avoided.

It is clear that a man of 170 lbs. weight has more proteid tissue to nourish than one of 130 lbs., and consequently what will suf for the latter will not for the former. Dietary standards are mer approximate and depend on the physical work to be performed, body weight, sex, age, climate, etc. There is doubtless a spec coefficient of nutrition characteristic of the individual.

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[1] Nutrition of Man.

[2] Physiologic Economy in Nutrition.

Chittenden demonstrated on professional men, soldiers, a athletes that they could perform their duties with greater vig and their strength as measured by tests, was increased under dim ished proteid diet; in fact, with about one-half the amount compared with the standards suggested.

CHITTENDEN'S TABLE

60 gm. of proteid are contained in—	Fuel value.[1] Calories.
½ lb. fresh lean beef (loin)	308
9 hens' eggs	720
⅘ lb. sweetbread	660
¾ lb. fresh liver	432
⅞ lb. lean smoked bacon	1820
¾ lb. halibut steak	423
½ lb. salt codfish (boneless)	245
2⅕ lbs. oysters, solid	506
½ lb. American pale cheese	1027
4 lbs. (2 quarts) of whole milk	1300
⅝ lb. uncooked oatmeal	1550
1¼ lbs. shredded wheat	2125
1 lb. uncooked macaroni	1665
1⅓ lbs. white wheat bread	1520
1¼ lbs. crackers	2381
1⅔ lbs. flaked rice	2807
⅗ lb. dried beans	963
1⅞ lbs. baked beans	1125
½ lb. dried peas	827
1 11/12 lbs. potato-chips	5728
⅔ lb. almonds	2020
⅖ lb. pine-nuts, pignolias	1138
1⅔ lbs. peanuts	3584
10 lbs. bananas (edible portion)	4600
10 lbs. grapes	4500
11 lbs. lettuce	990
15 lbs. prunes	5550
33 lbs. apples	9570

The standards of 100 gm. of proteid or more mean the *excret of excessive nitrogen* through the urine. Chittenden[2] found experiment that the average need of proteid food by adults is fu met by a daily metabolism equal to an exchange of 0.12 gm. nitrogen per kilogram of body weight. This means a catabolism ¾ gm. of proteid matter daily per kilogram. The intake of prot food must be somewhat in excess of proteid catabolism, since not of the proteid is available, and this is a variable amount depend on the proportion of animal and vegetable foods with their differ degrees of digestibility and availability. The required intake proteid Chittenden places at 0.85 gm. per kilogram of body weig giving a maximum for safety. Hence, for a man weighing 70 kil

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[1] Fuel value of the quantity needed to furnish 60 gm. of proteid.

[2] Physiologic Economy in Nutrition; Nutrition of Man.

(154 lbs.) there would be required daily 59.5 gm. (say, 60 gm.) of proteid food to meet the needs of the body. This is about one-half the Voit standard, and far below that of many other so-called diets. As the specialist is so often consulted as to the proper diet to maintain the health of the body, I quote Chittenden at some length, being a firm believer in his principles.

The daily proteid requirement of 60 gm. can be obtained from ½ pound of uncooked lean meat, of which loin steak is the type. Lamb, veal, poultry, or lean flesh of any variety, of equivalent weight, will approximately furnish the same amount of proteid.

Fish, such as halibut steak, and liver require ¾ pound, and of sweetbreads ⅘ pound are necessary.

Of salt codfish ½ pound is equivalent to the same weight of fresh beef, while of lean smoked bacon ⅞ pound is necessary.

Three hens' eggs furnish one-third the amount of proteid required in twenty-four hours. Dried peas and beans, almonds, and pine-nuts are as rich in proteids as the above-mentioned animal foods, and essentially the same weight is called for to provide the daily requirement of proteid. The same is true of cheese, the composition of ½ pound being equivalent to the same amount of proteid, but of much *higher calorie value* than the equivalent weight of fresh beef.

There are some differences in digestibility which tend to lower slightly the availability of the vegetable products, also of the cheese, which necessitates a slight increase in the amount of these foods to equal the proteid value of the equivalent weight of lean beef.

Certain foods are poor in proteids, such as fruits, bananas, grapes, prunes, apples, etc., lettuce, and, to a less degree, potatoes. These are all palatable, but add *little to the proteids*, even when given in large amount.

There is a radical difference between the animal foods and those of vegetable origin, in that the fuel value necessary to furnish the 60 gm. of proteid is small in the former, as compared with that of the vegetables; ½ pound of lean beef with its 60 gm. of proteid has, for example, a fuel value of only 308 calories, while ⅔ pound of almonds has one of 2020 calories; ½ pound of cheese has one of 1027 calories; ½ pound of dried peas, 827 calories. This is due to the proportion of fat or oil present. With fat meat, such as bacon, the calorie value rises in proportion to the increase of fat, the proteid decreasing to a greater or less degree.

A high proteid (animal) diet cannot serve for man. In a male, for example, with a weight of 70 kilos. and requiring 2800 calories, it would necessitate the ingestion of 4½ pounds of beef to secure this result, or nine times more proteid than is necessary for the system.

Certain vegetable foods on the diet list, such as flaked rice, crackers, and shredded wheat, contain proteids with carbohydrates and fat in such proportion that the energy requirement would be met with essentially by the same quantity as served to furnish the necessary proteid. In potatoes and bananas the fuel value pr dominates over the proteid. The ideal diet is an admixture, such wheat bread with butter or fat bacon to add to its calorie valu shredded wheat with cream, crackers with cheese, bread and mil eggs with bacon, meat with potatoes, etc.

Two quarts of milk will furnish half the requirement of an avera man, and reinforced by a 1-pound loaf of wheat bread, gives t requisite amount. A better combination is ¼ pound of lean bee ¾ pound of bread, and ½ pound of butter.

According to Chittenden, for a man of average weight of 70 kilo (154 pounds) to provide the requisite quantity of food—*i. e.*, 60 g of proteid and 2800 calories—the following is a sample dietary:

Breakfast:	Proteid. Grams.	Calories.
1 shredded wheat biscuit (30 gm.)	3.15	106
1 teacup of cream (120 gm.)	3.12	206
1 German water roll (57 gm.)	5.07	165
2 1-inch cubes of butter (38 gm.)	0.38	284
¾ cup of coffee (100 gm.) with	0.26	
¼ teacup of cream (30 gm.)	0.78	51
1 lump of sugar (10 gm.)	...	38
	12.76	850

Lunch:	Proteid. Grams.	Calories.
1 teacup home-made chicken soup (144 gm.)	5.25	60
1 Parker House roll (38 gm.)	3.38	110
2 1-inch cubes of butter (38 gm.)	0.38	284
1 slice lean bacon (10 gm.)	2.14	65
1 small baked potato (2 ounces—60 gm.)	1.53	55
1 rice croquette (90 gm.)	3.42	150
2 ounces maple syrup (60 gm.)	...	166
1 cup tea with 1 slice lemon		
1 lump sugar (10 gm.)	...	38
	16.10	928

Dinner:	Proteid. Grams.	Calories.
1 teacup cream of corn soup (130 gm.)	3.25	72
1 Parker House roll (38 gm.)	3.38	110
1 1-inch cube of butter (19 gm.)	0.19	142
1 small lamb chop broiled, lean meat (30 gm.)	8.51	92
1 teacup of mashed potato (167 gm.)	3.34	175
Apple-celery lettuce salad with mayonnaise dressing (50 gm.)	0.62	75
1 Boston cracker split, 2 in. in diameter (12 gm.)	1.32	47
½-inch cube American cheese (12 gm.)	3.35	50
½ teacup of bread pudding (85 gm.)	5.25	150
1 demi-tasse coffee		
1 lump sugar (10 gm.)	...	38
	29.21	951

The total with the dietary for the day amount to 58.07 gm. proteid and 2729 calories.

These figures are to be considered only approximately correct.

If a little more proteid is required without changing materiall the fuel value, a boiled egg can be added to the breakfast. A average egg of 53 gm. contains 6.9 gm. of proteid and increases the fuel value by 80 calories. If more vegetable proteid is desired, a soup of split peas can be introduced without changing to any great degree the calories; thus, one teacup of split pea soup (1.44 gm.) contains 8.64 gm. of proteid, while the fuel value is only 94 calories.

The addition of 1 banana (160 gm). will increase fuel value 153 calories, but will only add 2.28 gm. of proteid.

If the fuel value is to be increased without change in the proteid contents of the food, recourse can be had to butter, fat of meat, additional oil in salads, or to syrup and sugar.

Wheat products abounding in starch still show a large proportion of proteid; thus, shredded wheat biscuit (1 ounce), which is a type of many wheat preparations from bread and biscuit to various breakfast foods, yield about 3 gm. of proteid per ounce and 100 calories; ⅓ ounce of olive oil contains 100 calories. Potato, chiefly a carbohydrate, yields nitrogen the equivalent of about ¾ gm. of proteid per ounce. If a large volume is desired without much increase in real food value, there are green foods, such as lettuce, celery, greens of various sorts; fruits, such as apples, grapes, oranges, etc.

Meat augments largely the intake of proteid and adds relatively a small amount to the fuel value.

In edible nuts the content of proteid is high, in some cases higher than in fresh beef; while carbohydrates and fat are large in amount, as in almonds and peanuts.

In *pine-nuts and Brazil nuts carbohydrates* are small as compared with peanuts, almonds, and walnuts, an important fact where a vegetable rich in proteid is desired, but with freedom from starch (see table).

	Proteid present.	Carbohydrate present.	Fat present.	Water present.	Mineral present.	Fuel value per lb.
Almonds (edible portion)....	21.0	17.3	54.9	4.8	2.0	3030
Peanuts (edible portion).....	25.8	24.4	38.6	9.2	2.0	2560
Pine-nuts (edible portion)...	33.9	6.9	49.4	6.4	3.4	2845
Brazil nuts (edible portion)..	17.0	7.0	66.8	5.3	3.9	3265
Soft-shell walnuts...........	16.6	16.1	63.4	2.5	1.4	3285

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Green vegetables, such as spinach, help the bowel action.

Natural sugars are of value, especially such as occur in oranges, grapes, prunes, dates, plums, and bananas, and to a less degree in apricots, peaches, pears, apples, figs, strawberries, raspberries, and blueberries.

Apples when ripe and well masticated are good, and a baked apple is wholesome.

There is occasionally an idiosyncrasy to strawberries, and some suffer from fermentation from various fruits.

A diet conforming to true nutritive requirements must tend toward vegetable food if excess in proteids is to be avoided.

There is less need for food in hot weather, especially for fat, when lighter foods and less calories are required.

We must also remember that the excessive use of salt strains the kidneys.

The following table modified from that of Koenig and others gives the chemic composition of different foods and the heat units which they produce:

CHEMIC COMPOSITION OF COMMON FOOD SUBSTANCES

I. *Meats and Game.*

	Per cent. Nitrogenous (proteid).	Per cent. Fat.	Per cent. carbohydrate (nitrogen free).	Calories per 100.
Beef (very fat)	17.19	26.38	....	315.81
Beef (lean)	20.78	1.50	....	99.15
Veal (fat)	18.88	7.41	0.07	146.61
Veal (lean)	19 84	0.82	....	86.97
Mutton (fat)	14.80	36.39	0.05	399.31
Mutton (lean)	17.11	5.77	....	123.81
Pork (fat)	14.54	37.34	....	406.88
Pork (lean)	20 25	6.81	....	146.36
Westphalia ham	23.97	36.48	1.50	453.69
Salted ham	22.32	8.68	....	173.23
Smoked beef	27.10	15.35	....	253.76
Smoked beef tongue	24.31	31.61	....	393.64
Pulverized meat	64.5	5.24	2.28	322.53
Sweetbread	22.0	0.4	....	93.92
Chicken (fat)	18.49	9.34	1.20	167.58
Chicken (lean)	19.72	1.42	1.27	99.10
Capon	23.32	3.15	2.49	135.11
Duck (wild)	22.65	3.11	2.33	131.36
Partridge	25.26	1.43	....	116.85
Pigeon	22.14	1.00	0.76	100.02
Hare	23.34	1.13	0.19	107.08
Venison	19.77	1.92	1.42	105.44

II. *Fish.*

	Per cent. nitrogenous (proteid).	Per cent. fat.	Per cent. carbohydrate (nitrogen free).	Calories per 100.
Eel	12.83	28.37	0.53	312.93
Pike	18.34	0.51	0.63	83.57
Carp	20.61	1.09	....	94.64
Shellfish	17.09	9.34	....	156.93
Halibut	11.94	0.25	0.45	53.66
Salmon	15.01	6.42	2.85	132.93
Sardellen	22.30	2.21	0.45	113.83
Oysters	4.95	0.37	2.62	34.39
Fresh herring	10.11	7.11	....	106.15
Salt herring	18.90	16.89	1.57	247.61
Caviar	31.36	15.61	2.23	279.76

III. *Dairy Products*

	Per cent. nitrogenous (proteid).	Per cent. fat.	Per cent. carbohydrate (nitrogen free).	Calories per 100.
Cows' milk	3.41 to 4.3	3.0 to 3.8	3.7 to 4.81	56.41 to 71.93
Cream	3.61	26.75	3.52	276.01
Buttermilk	3.0 to 4.0	.93 to 1.3	3.0 to 4.0	33.08 to 43.63
Whey	.85	.23	3.03	18
Kumyss (cows' milk)	3.65	2.07	Lactic acid, .7 Alcohol, 1.9 Carbonic acid, 8.	32.99
Butter	0.5	90	0.5	823.1
Cheese (cream)	16.28	41.22	1.90	449.54
Cheese	34.99	11.37	5.40	269.06
Eggs (hens')	12.5	12.1	0.5	165
White of egg	12.67	0.25	....	54.22
Yolk of egg	16.24	31.75	0.12	355.99

IV. *Cereals and Vegetables*

	Per cent. nitrogenous (proteid).	Per cent. fat.	Per cent. carbohydrate (nitrogen free).	Calories per 100.
Wheat bread	6.0	0.75	52.0	245
Rye bread	6.11	0.43	46.0	217.56
Sago	0.5	traces	86.5	356.70
Wheat flour	8.5	1.25	73.0	345.78
Rye flour	10.0	2.0	69.0	342.50
Cakes	11.0	4.60	73.30	387.09
Roll	6.82	0.77	43.72	213.87
Zwieback	9.5 to 13.0	1.0 to 3.0	75.0	356 (average)
Cauliflower	2.0 to 5.0	0.4	4.0	35
Potatoes	1.5	....	20.0	88
Asparagus	2.0	0.3	2.5	20
Carrots	1.04	0.21	6.74	33.85
Rice	5.5	1.5	76.0	348.10
Beans	19.5	2.0	52.0	311.75
Peas	19.5	2.0	54.0	319.95
Spinach	2.49	.58	4.44	33.67
Oatmeal	12.05	5.26	66.77	338.80
Barley meal	8.31	0.81	75.19	323
Brussels sprouts	4.83	0.41	6.22	49.05
Cabbage (white)	1.89	0.20	4.87	29.52
Pickles	1.02	0.09	0.95	8.81

V. *Soups and Beverages*

	Per cent. nitrogenous proteid.	Per cent. fat.	Per cent. non-nitrogenous carbohydrate.	Calories per 100.
Meat broth	0.4	0.6	....	7.10
Meat juice (expressed)	6.0 to 7.0	0.5	0.5	31.20 (average)
Beef-tea	0.5	0.5		6.6
Leube's meat solution	9 to 11 albumin and 1.7 to 6.5 pepton.	....	....	86.5 (average)
Malt extract	8.0 to 10.0	....	55.0	258.30
Milk soup with wheat flour	5.0	3.25	15.0	112.
Barley soup	1.5	1.0	11.0	60.96
Rice pap with milk	8.8	3.5	28.6	182.61
Coffee	3.12	5.18	....	59.92
Tea	12.38	....	....	50.75
Beer	0.5	5.25	0.3	51.0
Porter	0.7	6.0	0.3	60.

VI. *Fruits, Nuts, and Sugar*

	Per cent. free acid.	Per cent. nitrogenous proteid.	Per cent. fat.	Chiefly sugar.	
				Per cent. non-nitrogenous carbohydrate.	Calories per 100.
Apples	0.82	....	....	7.22	29.6
Pears	0.20	....	....	8.24	33.78
Plums	1.50	....	....	4.68	19.18
Peaches	0.92	....	....	7.17	29.39
Apricots	1.16	....	....	4.69	19.22
Grapes	0.79	....	....	14.36	58.87
Strawberries	0.93	....	0.45	6.78	31.88
Chestnuts	....	5.48	1.37	38.34	192.11
Cane-sugar	....	0.35	....	93.33	382.65
Beet-sugar	....	....	....	99.75	408.97
Honey	....	1.20	....	73.22	305.22

For the determination of the calorie value of each kind of food the number of grams of albumin must be multiplied by 4.1, th grams of carbohydrate by 4.1, and the grams of fat by 9.3, and th multiples added will give the total calories as already described.

The following diet lists of von Noorden[1] demonstrate the metho of calculating calorie values, and will be found useful to fulfil thei indications:

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[1] Berl. Klinik, 1838, J. 55.

I. *A Chiefly Milk Diet with Addition of Carbohydrates in Liquid Form*

	Albumin (per cent.).	Fat (per cent.).	Carbohydrate (per cent.).	Calories per 100.
Milk, 1700 cc.	70.2	66.3	69.7	1295
Soup of tapioca flour, 30 gm. and 10 gm. albumose[1]	10	....	30	164
Soup of 40 gm. wheat flour, with some of the milk, 10 gm. sugar, and one egg	7.0	5.5	40	244
Total	87.2	71.8	139.7	1703

II. *A Chiefly Milk Diet with the Addition of Carbohydrates and Fat in Mushes and Soups*

	Albumin (per cent.).	Fat (per cent.).	Carbohydrates (per cent.).	Calories per 100.
Good milk, 1500 cc.	62	58.5	63	1056
Soup of 15 gm. sago, 10 gm. butter, 1 egg, 10 gm. albumose	17	13.5	15	257
Pap of 80 gm. corn flour, 1 egg, 10 gm. sugar (two meals)	7	5.5	90	398
Total	86	77.5	168	1711

III. *Milk Diet with Addition of Solid Food, Pastry, and Broths, leaving little Residue*

	Albumin (per cent.).	Fat (per cent.).	Carbohydrates (per cent.).	Calories per 100.
Milk, 1250 cc.	51	49	52	878
Meat broth with 1 egg, 10 gm. of butter, 50 gm. of fine toasted wheat bread (or softened)	10	14	30	294
Cakes 70 gm., butter 15 gm.	5	12	50	337
Soup of 30 gm. tapioca flour, 1 egg, 10 gm. butter	7	14	30	282
Total	73	89	162	1791

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[1] Ten gm. albumose is contained in 90 cc. (3 ounces) of Denayer's peptone preparation, in 22 gm. (℥vss) of Kemmerich's, or in 30 gm. (1 ounce) of Koch's.

IV. *Milk with Tender Meat, Solid Food (Pastry), Butter, and Soups*

	Albumin (per cent.).	Fat (per cent.).	Carbohydrates (per cent.).	Calories per 100.
Spring chicken, 100 gm.	19.6	2.8	....	106.4
Mashed potatoes, 100 gm.	2.0	4.0	20	127.4
Two eggs	14.1	11.0	....	160.1
Toasted wheat bread 100 gm.	7	0.5	55	258.8
Butter, 30 gm.	....	23.0	....	213.9
Trout, 100 gm.	19.3	2.1	....	106.4
Milk, 1250 cc. and soups in addition	51	49	52	878
Total	113.0	92.4	127	1851

V. *Abundant Non-irritating Diet*

	Albumin (per cent.).	Fat (per cent.).	Carbohydrates (per cent.).	Calories per 100.
Tender meat, 250 gm.[1]	49	7.0	....	266
Cacao, 20 gm.	4	6.0	8	105
Three eggs[2]	21	16.0	....	235
100 gm. Zwieback	8	1.0	75	349.4
100 gm. wheatbread	7	0.5	55	258.75
50 gm. cakes	4	2.3	36	187
50 gm. butter[3]	....	44.0	....	407
40 gm. tapioca flour[4]	....	....	40	164
40 gm. corn flour (maizena)	....	....	40	164
20 gm. sugar[5]	....	....	20	82
1250 cc. milk[6]	51	49	52	878
Total	144	125.8	326	3096.15

Digestibility of Food.—We may say that an article of diet i easily digestible if it makes small demand on the secretory and moto functions of the stomach, if it is readily absorbed, and causes n subjective disturbance. The scale of digestibility of foods has bee arranged according to the length of time that they remain in th stomach, the motor and secretory functions in the healthy stomac acting together, and hence the length of time the material remain in the organ indicates its digestibility; good motor power for a definit food shows good digestion. In pathologic conditions the rule i not as absolute, for either secretory or motor functions may b perverted alone or together, and sometimes in opposite directions. Gastric digestion may also be vicariously assumed by the intestines.

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[1] Meat of various kinds, finely chopped, raw, or broiled in butter, or roasted cold or hot, given in two meals.

[2] Egg in cocoa, one in soup and one raw or soft boiled.

[3] Butter for starchy foods, soup, etc.

[4] Tapioca flour to thicken soup.

[5] Sugar for cocoa and cornmeal pudding.

[6] Milk for cocoa, pudding, and to drink.

Leube's experiments were conducted with digestion in diseased stomachs, and from these he constructed his diet scale of foods according to their digestibility. It especially forms the basis of the diet of ulcer (Leube's method) and is valid in many other conditions.

Leube's Diet Scale.—*Diet 1.*—With much reduced digestion, the following are most easily digested: bouillon, meat solutions, such as Leube-Rosenthal's; milk; raw, soft-boiled, or poached eggs; zwieback; water, acidulous waters (Apollinaris, Seltzer).

Diet 2.—Less digestible are boiled calves' brain, boiled thymus, boiled chicken and pigeon, boiled calves' feet (with some); gruels, milk, mushes made with tapioca and beaten white of egg.

Diet 3.—If Diet 2 is digested, then these can be given: Raw beef (chopped fine), or scraped meat with a dull spoon, and roast meat scrapings in fresh butter; raw ham (chopped fine); a little mashed potato; stale white bread and a small amount of coffee or tea with milk.

Diet 4.—Roast chicken; roast pigeon; venison; partridge; roast beef, medium to raw (particularly cold); veal (leg); pickerel; boiled shad; macaroni; bouillon with rice. Trout are hard to digest. Small quantities of wine one or two hours before eating. Gravies are contra-indicated. Young and finely chopped spinach is best. Asparagus may be tried, but Leube considers it risky. After the fourth diet the food is gradually increased, but very gradually. Vegetables, salads, preserves, and fruits should be refrained from for a long time. A baked apple is one of the first of these that can be taken.

Penzoldt has formulated a scale of digestibility for the normal stomach.

Various Food Stuffs.—It is important to remember that we can replace the albumin in food by carbohydrates, and at times by the fats, though the latter are not always well tolerated. In many cases the food should be concentrated, nourishing, and finely divided, so as not to irritate the organ.

Milk is excellent in some cases, but in ulcer, too large quantities, at least 3 liters (1770 calories), would have to be given, which tend to overdistend the organ.

Moreover, raw milk remains in the stomach longer than some other forms of nutriment, and when given alone may coagulate, and with some disagrees. In some experiments at the Manhattan State Hospital,[1] in cases of dilatation of the stomach, it was conclusively shown that this organ emptied itself with greater rapidity in proportion to dilution of the milk with water. It is, therefore, preferable to give smaller quantities of milk and not to administer it alone, but diluted with barley-water, for example, or some other amylaceous material, such as rice-water or decoctions of tapioca, maizena, etc. One can add material, such as raw eggs, to increase the calorie value, a large amount of nutriment in small bulk, as described under the treatment of gastric ulcer. Gärtner's fat milk, from which some of the casein has been removed, is of service. Buttermilk has small nutritive value. Kefir, koumyss, bacillac, and matzoon are often useful.

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[1] Medical Record, June 20, 1908.

Meat broth and bouillon have little nutritive value, while beef-tea and expressed beef-juice are of some service. The latter is best made from the rump of beef, cut in dice, cooked for five to ten minutes, and then the beef-juice expressed and pepper and salt added. Valentine's beef-juice and Armour's extract of beef can be recommended and are cheap.

Gelatinous articles of food, such as gelatin in solution, jellies, calves' feet, etc., are easily digested, as are soups containing chicken or calves' brain finely macerated and forced through a sieve.

Shellfish, pike, halibut, and carp contain the least fat and are digestible.

Carbohydrates should be well masticated and carefully insalivated. If they contain much cellulose they are not as readily digested and should be ground thoroughly. If there is stagnation and so danger of fermentation, care in their use should be exercised.

Ordinary bread (rye or domestic) is not as good. Zwieback, toast, maizena, tapioca, oatmeal, and Löfflund's kindermehl are best.

Aleuronat flour (Ebstein) contains 80 per cent. albumin. Leguminose (Hartenstein's), Liebig's maltoleguminose, and Knorr's preparations are excellent. American veal has been found by experience to be not very digestible.

Fat in the form of butter (50 to 100 gm.) usually agrees.

Coffee and tea may be given in moderate amounts in many cases, but considerably diluted.

Alcohol.—Wines are recommended by many as a mild stimulant to the stomach in certain cases, but from personal experience I advise against their use. There are other remedies which give better results.

General Rules.—The teeth should be kept in good condition; thorough mastication and insalivation are important, and a brief period of rest after meals should be advised both in health and disease. Regularity of meals should be enjoined.

Method of Feeding to Spare the Stomach.—In some cases it is necessary to administer nutriment in soluble or easily digested form. Appended is a short list:

Hemmerich's pepton, somatose, sanatogen, tropon, Koch's pepton, Brand's meat preparations, Valentine's meat juice, Liebig's soup, and Gärtner's fat milk.

Leube-Rosenthal Meat Solution.—Chop 1 kilo. of beef into fine pieces, mixing it with water (1 liter), and add 20 gm. of pure hydrochloric acid and boil mixture ten to fifteen hours in a Papin pot.

The mass obtained is crushed, boiled fifteen hours longer, neutralized with pure sodium carbonate, and evaporated to a mushy consistency. It is digestible, but contains relatively small quantities of peptone.

Debove's Meat Powder.—Roast finely chopped lean beef on tin plates until it is entirely desiccated. Powder in a mortar.

Haggard.—Cut lean meat into narrow strips, place for a few minutes in hot fat or lard until surface is browned, then place on a sieve for a short time. Fat is allowed to drip off, and meat is dried twenty-four hours in a baking oven at a moderate temperature. It can be ground to powder in a coffee-mill.

Rectal Alimentation.—*General Rules.*—The bowels should be emptied by enema; injection of the nutritive enema should be given with the patient on the left side with a colon-tube several hours later; temperature should be about 100° F. (warm). A folded towel should be pressed against the anus and the buttocks pressed together for at least fifteen minutes after the injection.

Milk should always be peptonized, and alcohol, if injected, should not be stronger than 1:6 in the fluid enema. The addition of a small amount of salt aids absorption. Dextrose is readily absorbed. Occasionally it may be necessary to add a few drops of tincture of opium if there is much irritation. Raw eggs are readily absorbed. Sanatogen, pepton, and somatose are excellent additions. No more than 8 oz. (250 cc.) should be given at an injection. It can be administered four or five times a day. If the bowel is irritable, smaller quantities should be employed. The patient should remain quiet for one-half hour after the injection.

Ewald suggests 3 to 5 raw eggs, mixed with 150 cc. of water and 30 gm. of grape-sugar, and a small amount of common salt.

Boas uses 250 gm. milk; yolks of 2 eggs; salt, tablespoon of red wine; tablespoon of Kraftmehl.

I have found the following useful: milk (peptonized), 125 cc.; sanatogen, ʒiij (12.0); 2 raw eggs beaten up; water q. s., 250 cc. (℥viij), and a little salt. Modifications will readily suggest themselves.

Leube's Meat Pancreas is Often Valuable.—To 150 to 300 gm. of scraped and finely chopped beef add 50 to 100 gm. of pancreas from cow or hog, free from fat, and finely chopped.

The two substances are placed in a dish and 150 cc. of lukewarm water are added, and the mixture stirred till it forms a thick mushy mass.

If fat is also to be digested, add 25 to 50 gm. of fat. Inject with a pressure syringe.

Enemata of normal saline solution—ʒj (4.0) salt to Oj (500 cc.) of water—are of value for thirst, or administered at a temperature of 110° to 120° F. as a stimulant. Proctoclysis is useful. If this quantity is not retained, a smaller amount should be employed. Subcutaneous injection of fats I would not recommend. Sterile almond oil, ʒss (2 cc.), might be given three times daily subcutaneously in extreme emaciation for a brief period.

Inunctions with preparations of oil or lanolin may at times be of service.

DIET IN DISEASE

General principles will be enumerated.

In *acute diseases of the stomach*, as in acute gastritis, the indication is to spare the organ as much as possible. In some cases no food is administered by mouth for several days and rectal feeding is employed. If food is administered, it should be given in small quantities at first and in liquid form, weak broths, bouillon, barley-water, a small quantity of water. They should be neither excessively hot nor cold. Peptonized milk, milk and lime-water, white of raw egg beaten up; later, toasted bread, soft-boiled eggs, etc., and a gradual return to full diet.

Ulcer of the Stomach.—In this condition both mechanic and chemic irritation of the stomach should be avoided. Albumin solutions and finely divided proteid material are indicated, such as milk, beef-juice, somatose, tropon, or *especially, sanatogen* in solution.

Einhorn allows barley-, oatmeal-, or rice-water in early treatment. Raw eggs beaten up aid in binding the free acid. Dextrose and butter aid nutrition and lessen acidity. Starch in any great quantity is not well borne on account of the acidity.

Leube avoids all stomach feeding for several days after the hemorrhage, employing nutritive enema; while Lenhartz feeds immediately, binds the free acid, and endeavors to rapidly improve nutrition. *Gelatin solutions* are of value in these cases.

Chronic Diseases of the Stomach.—In the chronic diseases it is extremely important to see that sufficient quantity is taken and to improve the nutrition of the patient, as frequently subnutrition is present.

Carcinoma.—In malignant disease of the stomach or its orifices little can be accomplished by diet alone. The patient should receive frequent meals in small quantities, liquid or pultaceous in form. This is advisable, even if the cancer is so situated as not to interfere much with the motor function. I have seen cases improve temporarily in nutrition. Radical or palliative operation is indicated.

Benign Stricture.—Stricture of the cardia can at times be dilated with bougies; but with pyloric stricture perfect recovery often follows operation. With benign pyloric stenosis, hyperacidity, at times with hypersecretion, and ectasia are present. The indications in these cases are to administer liquid or mushy foods, chiefly of albuminous type. There is relative motor insufficiency and increased secretion. Starchy food in quantity is not, therefore, well borne.

The gastric *secretion*, *motility*, and *sensibility* of the stomach must be studied in every case.

When there are *disturbances of the motor function, food should be given so prepared* that it will be *most easily evacuated from the stomach, as in liquid or pultaceous form.*

Among the disturbances of gastric secretion we have hyperacidity and hypersecretion, an increase in the secretory function; and subacidity and anacidity, an impairment of secretion. Motor disturbances may complicate any of these conditions, and sensory disturbances are most frequent in the first class.

In the hyperacid forms, a diet should be selected which stimulates hydrochloric acid secretion as little as possible and at the same time combines with it. Abundant albuminous diet should be administered, with hyperacidity, in coarse form if the motor function is intact, otherwise in liquid or mushy form. With hypersecretion, smaller and more frequent meals are indicated, and fluids should be limited. Carbohydrates in any quantity are not well borne, though their digestion is better in hyperacidity than in hypersecretion. Solutions of dextrose are readily absorbed and lessen the secretion of hydrochloric acid. The diet of ulcer has been previously referred to.

Fats are quite well borne, and in the form of olive oil lessen the hydrochloric acid.

In cases of subacidity, as in chronic gastritis, meat must be diminished and carbohydrate material increased. Nutrition must be improved. Koumyss, matzoon, milk, and raw eggs must be taken with crackers and butter between meals.

As an example of anacidity we have achylia gastrica. This may be a temporary functional disturbance or a permanent condition with organic changes. Chemic action has ceased and vegetable food (in which starch granules possess an albuminous coat) as well as animal food pass from the stomach unchanged and irritate the intestines. A rich carbohydrate diet is indicated, but it must be well pulverized.

With ectasia and gastroptosis small and frequent meals are indicated, the character depending on the gastric secretion. With severer forms of motor insufficiency liquids and mushes are required.

In nervous dyspepsia one must gradually increase the quantity of food. The administration of frequent small meals, koumyss, matzoon, etc., between the chief meals is of value. The rest cure is often serviceable.

One must individualize in every patient, and with the aid of the general principles described it will be possible to formulate a diet to suit the case.

CHAPTER VIII

LOCAL TREATMENT OF THE STOMACH

LAVAGE

SINCE the year 1867, when Kussmaul employed lavage in a scientific manner with his stomach-pump in the treatment of diseases of the stomach, there has been a gradual improvement in the type of instrument. The hard tube and the use of the stylet for introduction have passed into disuse, and the modern method is based upon the principle of siphonage with the soft-rubber tube.

Funnel Method.—The one that is in most common use for washing the stomach is by means of the funnel. The latter may be of hard rubber, glass, or a flexible rubber funnel, attached to a piece of soft-rubber tubing about a yard long, the latter being joined to the upper end of the stomach-tube by a small connecting glass or hard-rubber tube (Fig. 73).

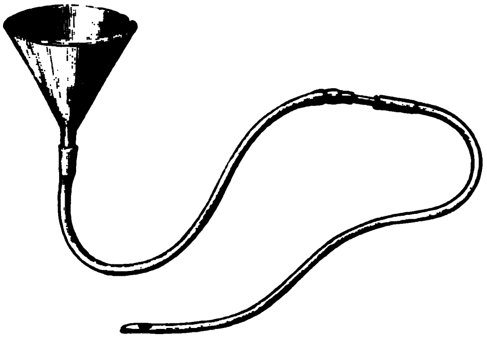

Fig. 73.—Funnel and tube for lavage.

The glass funnel is more readily broken and the soft-rubber funnel not as easily managed by a novice, so that for general use the hard-rubber funnel is preferable. One of medium size, holding about 250 cc., is most convenient. The glass connecting tube, in one case to my knowledge, came near being the source of considerable danger to the patient. During the lavage he suddenly grasped the glass tube and managed to splinter off a portion of it, fortunately at the same time partially pulling out the stomach-tube. The accident was immediately noted and the tube entirely withdrawn. Fragments of glass were found in the tube.

There is always the possibility of a similar accident to the glass funnel, and the use of a rubber instrument and metal or rubber attachment would seem to be preferable.

Dangers.—There is one possible danger, namely, the stomach-tube may separate from the attachment to the funnel tube and slip down into the stomach. Leube[1] reports such a case. On the ninth day after swallowing the tube—after an attack of coughing—the tube passed up into the esophagus and pharynx and was withdrawn therefrom. The connection between the stomach-tube and funnel-tube had become loosened and the water from the latter forced the tube into the mouth.

Moreover, every stomach-tube *should be tested before using* to be sure that it is not cracked or weakened by some defect.

Friedenwald[2] reports such an accident resulting from the use of a defective tube, with the result that two fragments were subsequently removed by gastrotomy.

At the Manhattan State Hospital, Ward's Island, among our nervous and insane patients, a long, continuous, single-piece stomach-tube (3½ to 4½ feet in length), with a rubber funnel at the end, is employed. This obviates all possible danger of swallowing the tube, which would be the most likely accident with this class of patients.

For ambulance work such a tube is decidedly to be recommended.

Under ordinary conditions, with the two-piece tube, the patient or *an assistant* can hold the stomach-tube tightly at the point of junction with the connecting joint, or the operator can hold it firmly at the lips of the patient when he elevates the funnel.

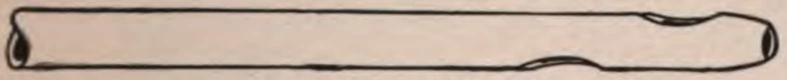

Fig. 74.—Best tube for lavage.

Selection of the Stomach-tube.—The selection of the stomach-tube may seem to be an unimportant matter, but from a varied experience there is undoubtedly a decided advantage in the choice of an instrument.

The tube should be of value both for aspiration of the stomach contents and for lavage. Some prefer the tube closed at the end and with two lateral openings, claiming that there is less danger of traumatism from the smooth rounded end and less chance of aspirating the mucous membrane into the openings of the tube.

In actual practice, one has to exercise more pressure with this type of tube in order to force it to take the curvature of the stomach and lie parallel with the same, if thorough aspiration or lavage is to be performed. This tube is, moreover, not so readily cleansed.

The tube *open at the end* and with a large lateral opening is preferable; a tube with two lateral openings is the best, though not absolutely essential (Fig. 74).

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[1] Deutsch. Arch. f. klin. Med., vol. xxxiii, p. 6.

[2] American Medicine, August 2, 1902.

It is much easier to thoroughly aspirate the stomach or perform lavage with tubes of this description. The pressure is so minimized by these large openings that in my own experience I have never seen damage result to the mucous membrane.

For practical purposes a tube of from 28 to 30 (French) is thoroughly efficient. I have seen physicians in their general practice employ tubes of very large caliber, irrespective of the size or age of the patient. This is about as sensible as using a large sound in every case, irrespective of caliber or conditions of the urethra.

The tube of medium caliber that I have indicated will pass comfortably through any average or even small adult esophagus, unless stricture be present. The best tubes are marked at about 18 inches from the distal end (it is 16 inches from the teeth to the stomach), and by this means we know when the instrument has reached that organ. The tube should be inserted a little further until checked by the lower border of the stomach, and then withdrawn slightly until no resistance is felt by the operator. With ectasia or gastroptosis it may be necessary to pass it a considerable distance.

If the tube is not marked it is easy to estimate the correct distance and make a scratch mark on the tube with a pencil, or insert it until checked by the lower border of stomach, as just described.

A pitcher, pail or large basin, towels, and rubber sheets are required. Special irrigating stands can be secured, but are unnecessary and expensive for the young physician.

The correct method of lavage in office practice, without an assistant, is first described. *False teeth should be removed.*

The patient should sit upright in a straight back chair, with a rubber sheet or towels about the neck and protecting the front of the body. His confidence should be gained. He should be told that the procedure is slightly disagreeable, but absolutely safe. He should be admonished that he is to breathe deeply and steadily all the time, as this will prevent the gagging and sensation of choking; and he should be directed, while the lavage is actually in process, to follow out this method.

The stomach-tube should be lubricated by dipping it in warm water. Vaselin, cold cream, or olive oil may be used, but they are disagreeable and unnecessary. The irrigating fluid should be pleasantly warm to the hand. This is sufficient index to the temperature, though for absolute accuracy a thermometer may be employed; and about 100° to 101° F. is correct.

Plain water or, preferably, normal saline solution—ʒj (4.0) salt to 1 pint (500 cc.) of water—is employed in the average case for the purpose of cleansing the stomach. Special solutions will be appropriately indicated in their places.

The patient should bend the head slightly forward and open the mouth, but not protrude the tongue.

In rare cases a 2 per cent. cocain or 5 per cent. eucain spray may be required if the pharynx is irritable. Freezing the stomach-tube is also serviceable.

The physician should never insert his finger into the mouth to depress the tongue or act as a guide to the tube, as it renders the operation more difficult, and will only cause gagging or vomiting.

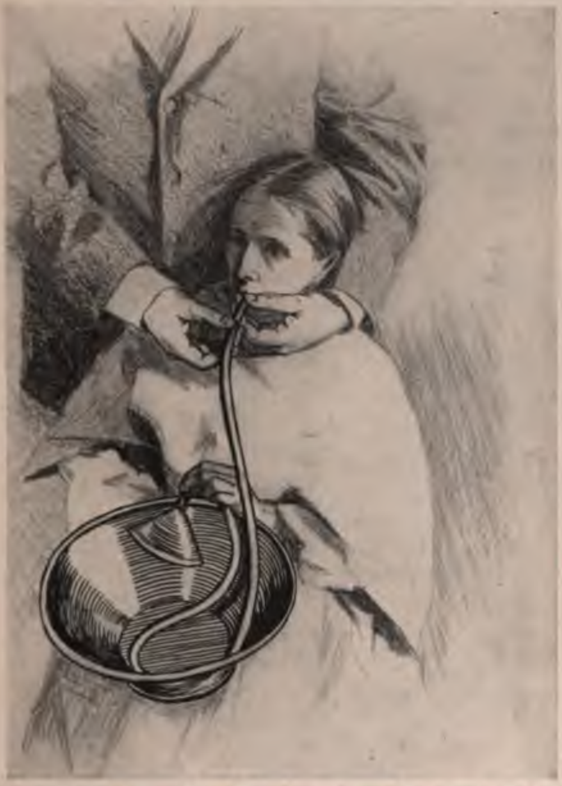

Fig. 75.—Correct method of passing the stomach-tube.

Most of our text-books advise that the operator stand in front of the patient and pass the tube along the base of the tongue.

The most practical method is the one shown in Fig. 75.

The physician stands on the right side and a little back of the patient and passes the left arm about the neck, the fingers *supporting the tube at the lips*, the little finger resting on the chin.

This method prevents the patient from throwing his head back and struggling, and gives the operator perfect control.

The stomach-tube should be passed into the mouth with the right hand, it being held about 2 inches from the lips and being supported by the left hand against the roof of the mouth. It shoul then be rapidly forced in with the right hand, the index-finger an thumb of the left hand continuously aiding its introduction.

The tube follows the arch of the mouth, and glides down th posterior wall of the pharynx (Fig. 76). *Interference from the tongu* is thus *avoided.*

When its progress is checked, the patient should be told t swallow and it will enter the esophagus. It should now be rapidl fed into the mouth until the marked ring has been reached, an then more slowly to the bottom of the stomach.

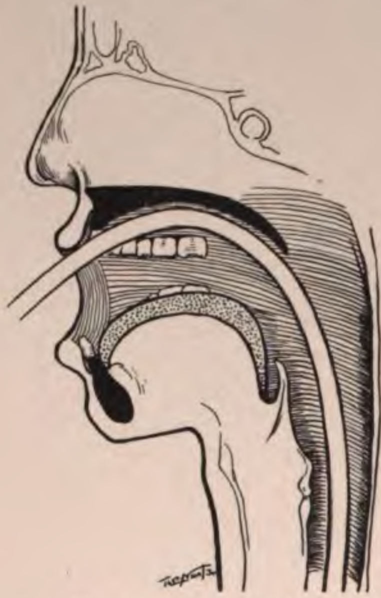

Fig. 76.—Course of tube.

If the tube is checked durin introduction, it is probably du to a spasm of the esophagus, an the act of swallowing or a dee inspiration will free it. There i practically no danger of its en trance into the larynx.

Sometimes the patient ma become cyanotic and a beginne may fear that this has happened but this is due to the fact tha the breath is held, and deep an regular breathing will immediately relieve the condition.

Occasionally the tube ma slip out of the esophagus an coil in the mouth, but that i easily detected. Patients accustomed to lavage can often introduce the tube themselves.

I prefer a funnel[1] of a capacity of 250 cc. or about 8 ounces, and allow 2 funnelfuls to run into the stomach (in all about 1 pint) and to run out again at once. It is a bad practice to overdistend the stomach, just as it is the bladder. At times I employ 1 funnelful.

Lavage is continued until the water is perfectly clear. The patient may move the body about so as to bring the water in more thorough contact with the stomach wall, or may lie down, as suggested by Fleiner, rotating to the right side and then to the left; but this is rarely necessary except in cases of marked dilatation with insufficiency.

Lavage by a Single Operator.—During lavage the patient steadies the stomach-tube, holding it to the lips with one hand. He should be instructed not to bite the tube.

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[1] If the funnel is of smaller capacity, more funnelfuls should be used, total 250 to 500 cc. (8 ounces to 1 pint).

The operator holds the funnel in the left hand and pours the water into it from a pitcher in the right hand, then elevates the funnel to about the level of the patient's forehead (Fig. 77).

As soon as it is empty, he pours in the second funnelful. Then before the latter is empty he quickly lowers it to below the level of the patient's stomach, and allows the fluid to siphon out into a pail or bowl placed on the floor to the right of the patient (Fig. 78).

The rim of the funnel should be held upward. It should be allowed to fill before emptying it, as in this way it can be estimated whether the amount of fluid that flows out equals that which was poured in. In emptying the funnel it should only be slightly tipped, so that the column of water is still visible in the bottom. This prevents the entrance of air and also the aspiration of the mucous membrane into the openings of the tube.

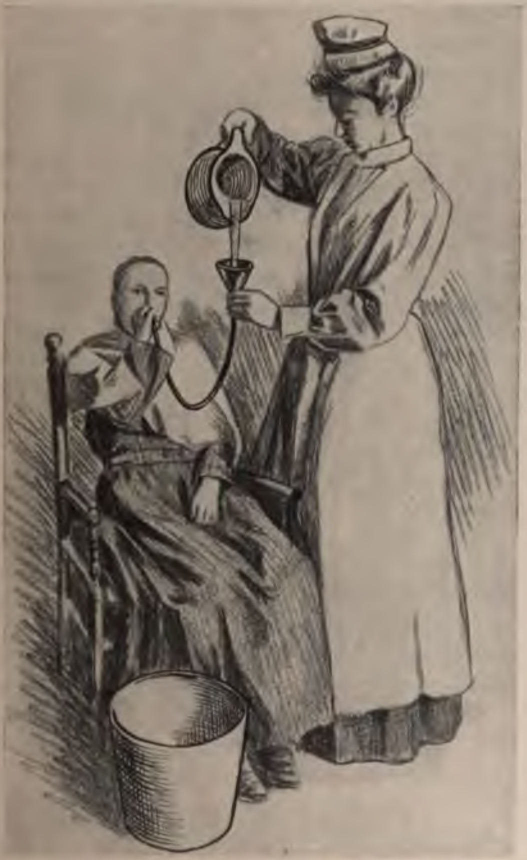

Fig. 77.—Lavage by single operator: Position one.

When the washing is completed, the funnel should be rapidly

raised and the stomach-tube withdrawn in this position. The sm
column of water remaining in the tube flows back into the stoma
and prevents the possibility of the accident just noted. The tu
should not be pinched during the withdrawal, as mucous membra
might be aspirated in.

If the water stops flowing during lavage, one should observe
the patient has not inadvertently withdrawn the tube slightly; a
in this event, it should
pushed back.

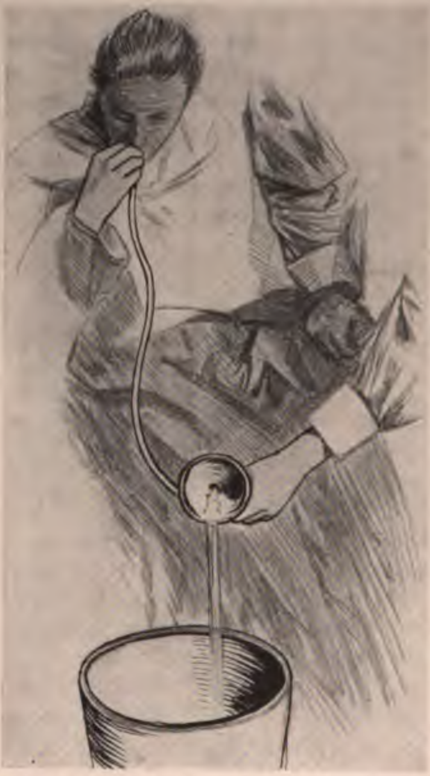

Fig. 78.—Lavage by single operator: Position two.

On the other hand, t
tube may be bent and the fl
stopped by reason of its bei
forced too far into the stomac
If so, withdrawing it slight
will correct the trouble.

Pieces of food may occlu
the tube by stopping up t
stomach openings. By pou
ing more water into the funn
the instrument will usually
cleared out. By forcing a
through the stomach-tube
by suction this can be accon
plished if the above meth
fails. Thus, a rubber bu
with metal attachments is i
serted between the stomac
tube and the funnel-tube,
place of the tube joining the
Close the end leading to t
funnel and compress the bul
This will force air through t
stomach-tube; or first con
press the bulb, then close t
distal end connecting with t
funnel by making an angle
compressing the soft tube
the latter then let the rubber bulb expand again. The last meth
described is that originated by Friedlieb. The stiff rubber bu
made by Tiemann, with metal attachments, is a better instrume
than Friedlieb's, which has glass attachments.

On rare occasions it may be necessary to remove the tube, clea
it, and reintroduce it.

In emergency in country practice one can employ an ordina
tin kitchen funnel and a piece of small red or even white rubb
tubing. A fountain syringe with the long soft-rubber tubing h
been used in poison cases. A stomach-tube can be improvised o

of a long colon-tube. The lateral opening can be cut with a pair of sharp scissors, and the rough edges of the window burned over an alcohol lamp, wiping them quickly with a wet cloth, thus making a smooth velvet eye. If the opening of the improvised tube is rough, it can be trimmed down and smoothed by the above method.

It is best to lubricate all such emergency tubes with olive oil or vaselin, as they are less smooth and more difficult of introduction.

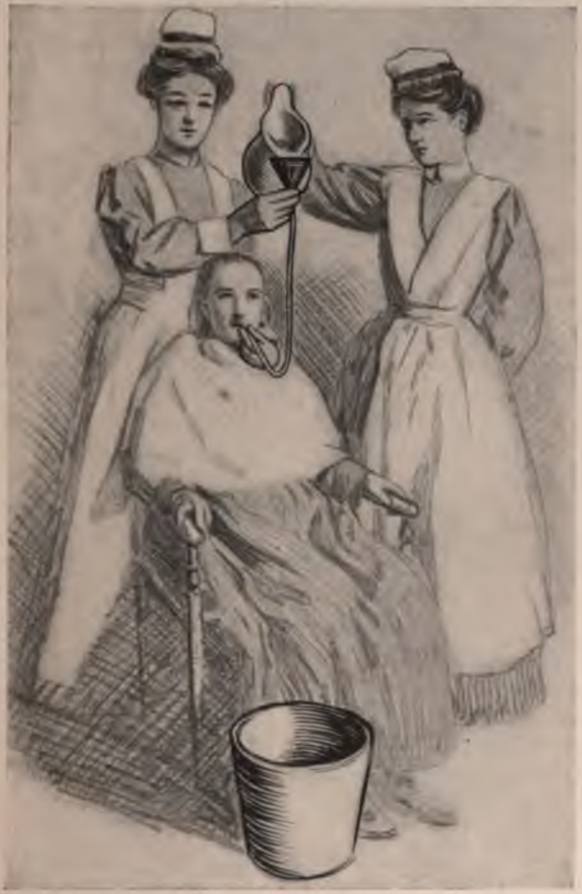

Fig. 79.—Lavage by two nurses: Step one.

With an intelligent patient who will co-operate with the physician the method described is excellent. In nervous cases, when possible, it is easier to have an assistant, or for two nurses to perform lavage, as in Figs. 79 and 80.

The technic is the same as to passage of the tube, etc. One nurse steadies the tube at the patient's mouth and carries on the manipulation with the funnel, the other pouring the fluid.

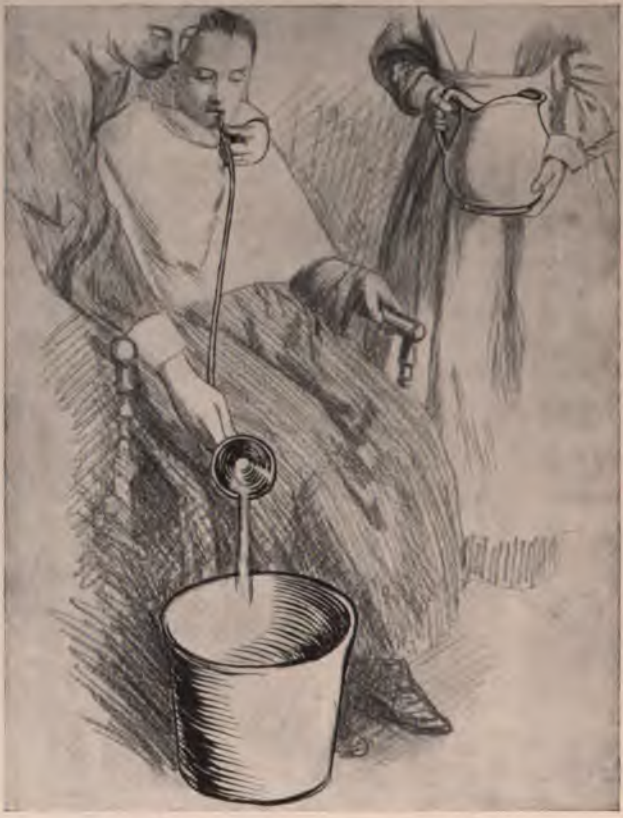

Fig. 80.—Lavage by two nurses: Step two.

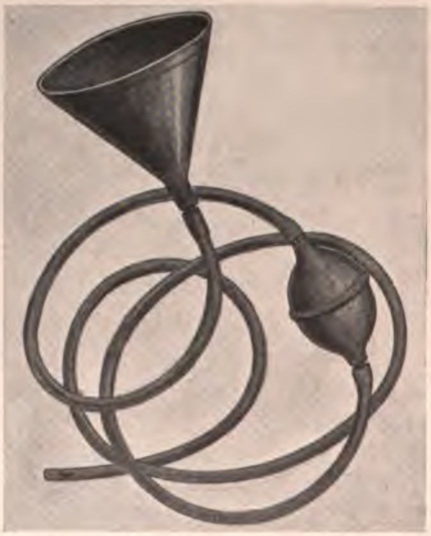

Fig. 81.—Friedlieb's apparatus for lavage.

If the patient resist, nurse No. 1 both holds the tube and prevents interference by the patient's hands, while nurse No. 2 manages the funnel and pours the fluid. Friedlieb's apparatus is demonstrated in Fig. 81.

In Figs. 82 and 83 we have two nurses performing lavage with a funnel, the *modified Friedlieb bulb* connecting the stomach-tube and funnel-tube. This is a favorite method of mine.

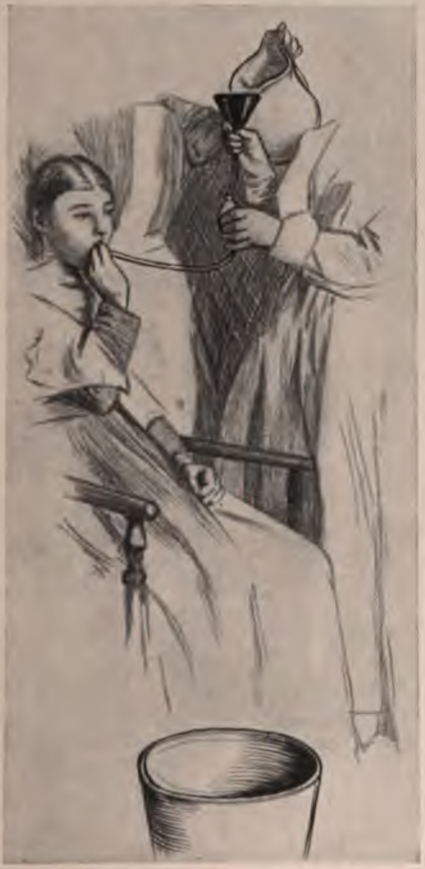

Fig. 82.—Nurses performing lavage with modified Friedlieb bulb: Step one.

It is best to close the bulb on the side joining the stomach-tube by pinching the latter with the fingers, then squeeze out the air and pour the fluid into the funnel, allowing the bulb to fill with water. This prevents aspirating air into the stomach. Lavage is then performed in the usual way. The advantage is that we *have the bulb in position to employ air pressure or suction* if the stomach-tube becomes occluded. Intermittent squeezing of the bulb also aids expulsion of the fluid.

The modified bulb made by Tiemann has metal connectio (instead of glass) and is stiffer than Friedlieb's.

Irrigation of the Stomach by Means of a Glass Y or T.- This method has generally been known as Leube-Rosenthal's, b R. H. M. Dawbarn has taught this plan for many years. It useful for office work.

A large glass irrigator, about 2-quarts capacity, is hung at level slightly above the patient's head. This should be marked

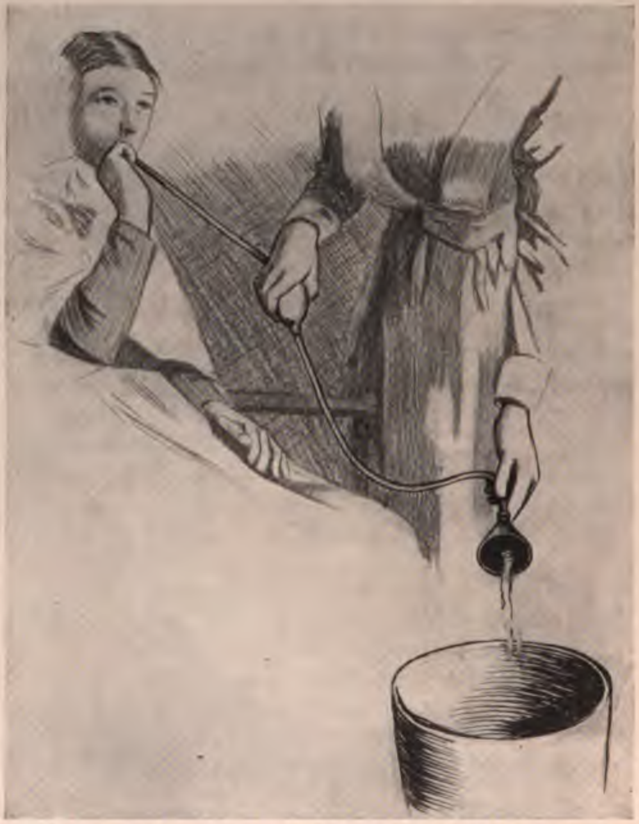

Fig. 83.—Nurses performing lavage with modified Friedlieb bulb: Step two.

250 to 2000 cc.'s, or in ounces or pints. This irrigator is co nected with a long soft-rubber tube by means of a Y- or T-shape glass or, *preferably, hard-rubber or metal* tube; one branch with th stomach-tube and the other with a carry-off tube which passes dow into a pail or basin.

The irrigator tube should be *closed with a clamp.* This is ke closed until after the introduction of the stomach-tube and co mencement of lavage. A second clamp on the outflow tube *unnecessary*, as all manipulation can be thereafter conducted by the operator, by alternately opening and closing the inflow and outflow tubes with the fingers.

The stomach-tube is introduced in the way described. The outflow is pinched as in Fig. 84 and the clamp on the inflow tube opened. Only 500 cc. of fluid, or about 1 pint, is allowed to flow into the stomach. I do not approve of employing the larger quantities as recommended by some authors.

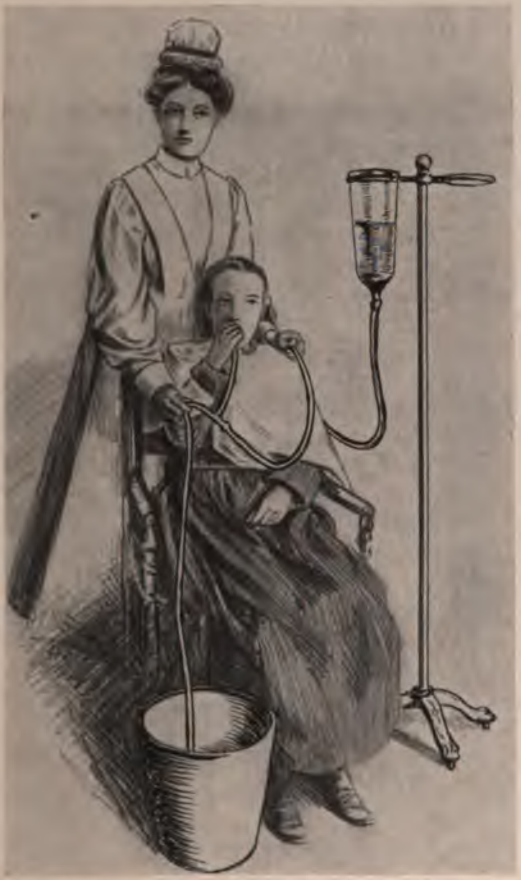

Fig. 84.—Stomach irrigation by the T-method: First step.

While the fluid is still entering, the outflow tube is suddenly released and part of the current is diverted, thus starting the siphon action. The inflow tube is then pinched, as in Fig. 85, and the stomach rapidly empties itself. The outflow is then pinched and the inflow released, and so on. The patient may shake his abdomen, so as to wash all parts more thoroughly.

This procedure must be continued until the wash-water retu clear.

There are cases among the insane or when the patient is unc scious when the methods described cannot be used.

It may be necessary to *employ a mouth-gag*, forcibly distend jaws, and force in the stomach-tube. Under such conditions num ous assistants may be required.

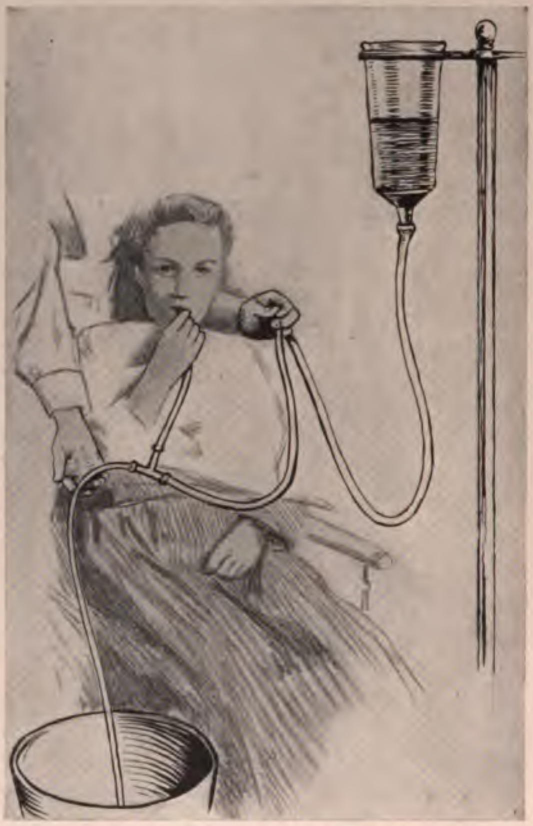

Fig. 85.—T-method: Second step.

The operation may be performed with the patient lying on back, being properly restrained. The tube should be passed al the roof of the mouth, and the patient should lie on a sufficier high level, so that the funnel can be carried below the level of body in order to secure the siphon effect.

If no mouth-gag can be secured, the handle of a spoon, protec with gauze or a handkerchief, can be inserted between the te turned, and the jaws forced apart, or some such instrument improvi

The director devised by Mark Knapp (Fig. 86) would be of va in such cases. Being all metal, it can be left *in situ* during lavage, and acts as a gag.

The simplest method in such cases is to introduce the tube through the nostril.

The technic of lavage by this method is shown in Figs. 87 and 88.

The tube is passed along the floor of the nostril, just as is the guide for posterior nasal tamponnade. It is fed directly through the nostril for about 20 inches, and no difficulty, as a rule, is experienced, as it passes down the posterior wall of the pharynx and so on into the esophagus.

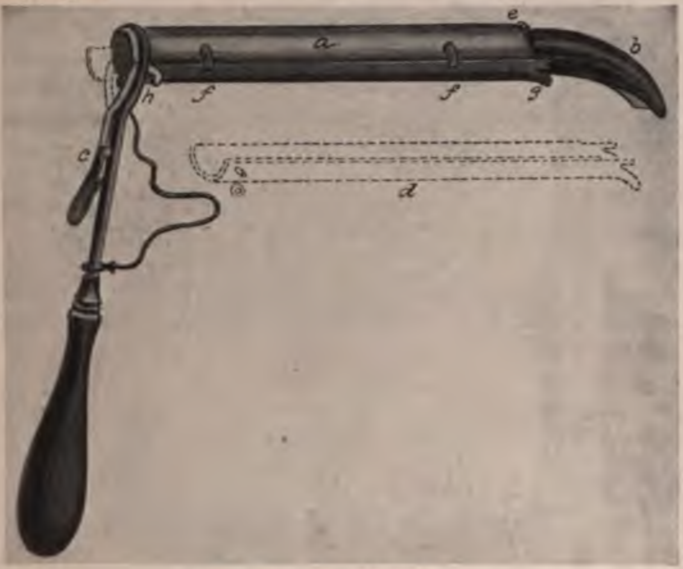

Fig. 86.—Knapp's director.

The nares should be examined, and that nostril selected which is of larger dimensions. This is important, as there are frequently deflections of the septum.

At the Manhattan State Hospital a stomach-tube of fair size (about 27, French) is employed. I frequently use this tube in my office lavage per orem. It is rarely necessary to employ the very small tubes so frequently recommended. For thorough lavage a fair caliber is preferable.

Gavage is a different proposition and the small tubes are to be preferred.[1]

Hemmeter and others have employed double-current tubes for lavage, but I can see no advantage.

Indications for Lavage.—1. *In All Cases of Poisoning.*—Some authors advise against it in cases of acids or alkalis, for fear of causing perforation; but there is greater danger of the latter by leaving the poison, since thorough emesis cannot be secured, especially if the patient be unconscious; there is the increased danger of subsequent damage to the intestinal canal below and often to the other organs,

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[1] Gavage or forced feeding is employed chiefly among the insane who refuse food, the tube being introduced as in lavage, the *nasal route* being preferable.

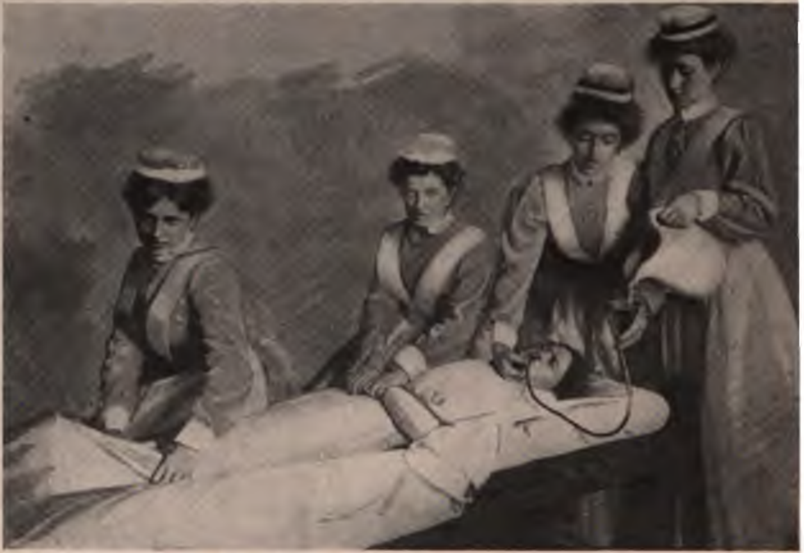

Fig. 87.—Lavage through the nostril: First step.

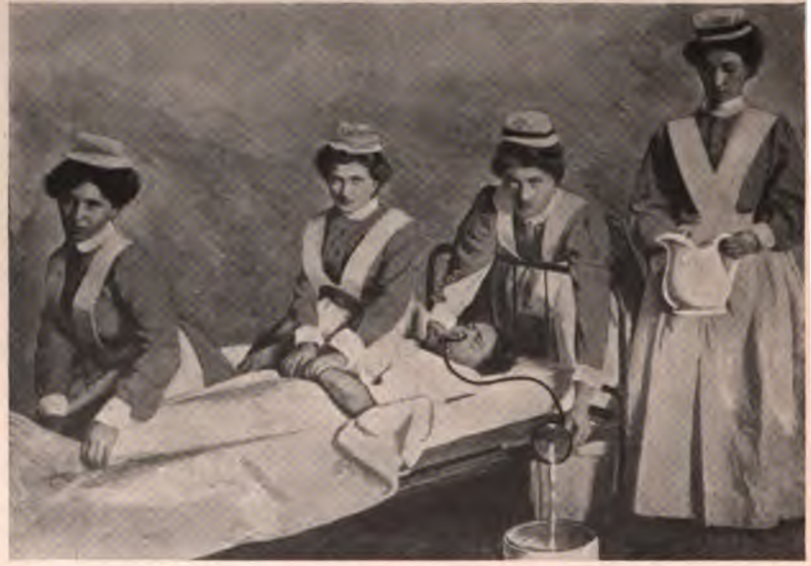

Fig. 88.—Lavage through the nostril: Second step.

such as the liver and kidneys, or cardiac or respiratory poisoning might result.

2. In *acute* and *uncontrollable vomiting* from any cause, as from acute gastritis, the value of lavage was first definitely shown by our specialists in pediatrics. Bilious vomiting is included.

3. In *chronic gastritis*, with excessive production of mucus.

4. In *dilatation of the stomach* (atonic type), where there is marked fermentation and motor insufficiency.

5. In *dilatation of the stomach* (stenotic type), with fermentation, motor insufficiency, gastritis, etc.

6. In *acute dilatation of the stomach* from all causes.

7. In *vomiting due to vicious circle*, after gastro-enterostomy.

8. *At the end of anesthesia*, to prevent post-operative vomiting, or to treat the same, if it has occurred.

9. In *post-operative intestinal paresis* (correctly, gastro-intestinal paresis) lavage should be employed together with enteroclysis.

10. In *acute tympanitis of typhoid fever* lavage is of great value to aid reduction of the same; especially when hemorrhage is occurring and enteroclysis is contra-indicated.

11. In *intestinal obstruction*, especially in intussusception. Frequent lavage has so relieved abdominal distention above the point of obstruction that the condition has been spontaneously reduced. It also checks the vomiting in this condition.

12. Occasionally lavage with iced water has proved successful in *uncontrollable hemorrhage from gastric ulcer*. It should be used as a last resort and with great caution.

13. It is also of service in *estimating the degree of motor insufficiency* by washing out the residue after aspiration following the test meal.

14. In *gastric tetany*.

15. In the *convulsions of young children* which immediately follow the ingestion of improper food. Epileptiform convulsions occasionally occur in adults from overfeeding and lavage is indicated.

16. In *vomiting of peritonitis*.

Contra-indications to Lavage.—1. *Aneurysm of the aorta.*

2. *Gastric hemorrhage*, as a general rule, except as in No. 12 above.

3. *Marked heart lesions*, when danger might be incurred, such as in angina, etc.

4. *Last months of pregnancy.*

5. *Special conditions* where it might damage the patient or be dangerous.

In *cases of poisoning* lavage would take precedence over all other risks.

6. *Recent rectal*, *vesical*, or *renal hemorrhage*.

7. *Recent hemoptysis.*

8. *Extreme prostration* from any cause.

Stomach Douche.—This method was first described by Malbranc, and the measure was first employed by Kussmaul. It consists in sprinkling the stomach with water under pressure. Ewald and Rosenheim[1] have devised the most practical stomach-tubes for this purpose.

The instrument, as in Fig. 89, has numerous small lateral openings and a slightly larger hole at the end. This last is so that the water can run off more rapidly in emptying the stomach, and any mucus or food products can more readily escape. If the hole is too large, the bulk of the fluid will pass through this and no diffuse irrigation of the mucous membrane be accomplished.

The tube is introduced in the manner already described, and the funnel method is employed. This is held quite high above the patient, so that the water is under considerable pressure and numerous small streams are forced out of the tube.

Rosenheim recommends it on an empty stomach before breakfast or, if this is impractical, three or four hours after the first meal. It is applicable to mild motor insufficiency. Rosenheim[2] advises its use in mild cases of chronic catarrh and in irritation of the sensory and secretory apparatus.

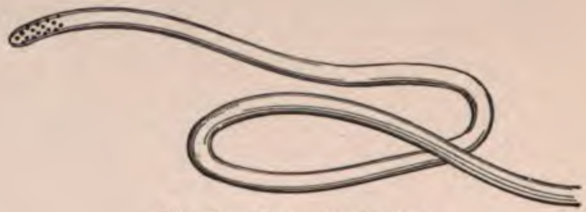

Fig. 89.—Stomach douche.

Salt added to the irrigation fluid increases the hydrochloric acid production, nitrate of silver (in 1 : 1000 strength) reduces the secretion of gastric juice. Other observers have agreed with these findings, and Riegel advocates the use of the silver nitrate. Fleiner[3] recommends the douche for stimulating the appetite. Thus, infusions of hops, quassia, condurango, or cinchona bark are valuable.

Boracic acid, salicylic acid, sodium salicylate, thymol, gomenol, creolin, lysol, etc., may be employed in average strength (1 : 1000) if antiseptic treatment is indicated.

If medicated douches are employed, the stomach should first be washed with plain water; then the medicated fluid applied for one to three minutes and then siphoned out; the stomach should be rewashed with plain water if toxic materials have been used.

Gross has devised a *double-current gastric douche*, which is scarcely practical, and Einhorn an instrument with a ball-valve and hard-rubber tip.

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[1] Therapeut. Monatsch., August, 1892.

[2] Berlin. Klinik, 1894, No. 71.

[3] Samml. klin. Vorträge, New Series, No. 103.

The following simple addition to the Ewald-Rosenheim tube gives satisfactory results. Employ a fountain syringe instead of a funnel and also a compression bulb (Fig. 90).

The stomach douche is passed in the usual way and the bulb (aspirating) joins it to the fountain syringe, which contains the irrigating fluid at a temperature of 100° F. Clip X is previously closed. The tube is pinched tightly at *A* and the clip then opened. By squeezing the bulb *B*, all air is driven out through the fountain syringe *C*. The bulb is then released and fills with fluid. This prevents air from entering the stomach. The finger releases the tube at *A* and the fluid begins to flow into the stomach; and by intermittent pressure of bulb (*B*) the spraying effect can be intensified at the will of the operator. When the douching is completed the bulb is detached from the fountain syringe and with pressure of the same—the thumb over the end—slight aspiration is commenced and the contents will then siphon out.

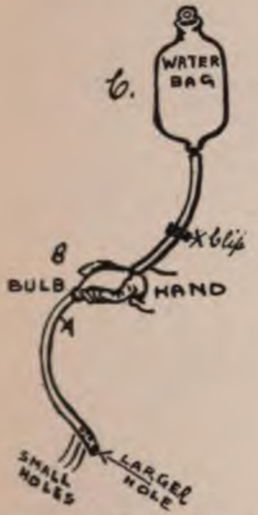

Fig. 90.—The bulb-compression method of spraying the stomach.

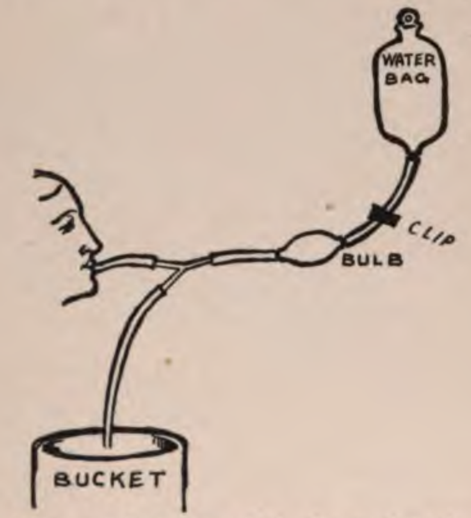

Fig. 91.—The Y-method of spraying the stomach.

In Fig. 91 is illustrated the Y-method of spraying the stomach. The technic is the same as the similar procedure in lavage.

In Fig. 92 the use of the alternate hot and cold douche, with pressure bulb, is depicted.

The addition of the bulb enables one to employ the spray without distending the stomach with air, and to reach *much further than with simple hydrostatic pressure*. When stronger medicaments are employed, the rapid emptying of the stomach within one to two minutes and immediate lavage eliminate all danger, especially in view of the fact that by the compression method of spraying much less fluid is required, as compared with the older procedures.

The temperature of the fluid should be about 100° F. When stimulating effects are required it could be 103° to 105° F.; if cold, at 75° to 80° F. when used with the alternate douche.

In atony, with or without commencing dilatation, the spray method, employing in all not over ½ pint to 1½ pints, is at times of service to stimulate the organ. With this exception, I rarely use it.

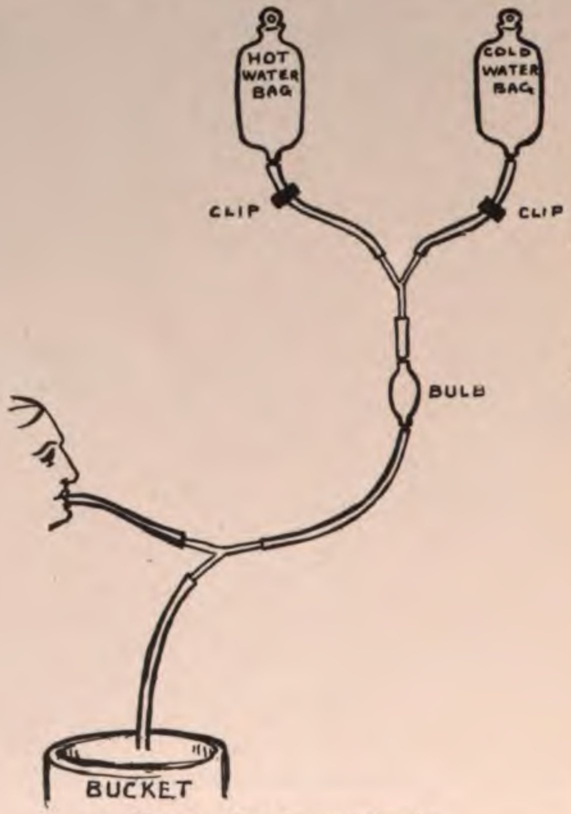

Fig. 92.—The alternate douche.

Gastric Spray.—This consists of an ordinary spray apparatus with a double bulb, to which is attached a soft Nélaton tube 70 cm. long; within this is a fine central flexible tube, which connects the inner capillary tube with the nozzle (Fig. 93).

Einhorn[1], who devised the tube, recommends it highly, as thereby one employs a small amount of fluid to secure results; and hence, there is less danger when toxic or irritating substances are used.

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[1] New York Medical Journal, Sept. 17, 1892.

The spray should be employed when the patient has fasted or after a previous lavage. The bottle is filled with the required amount of the antiseptic solution, the tube dipped in warm water, and introduced in the usual manner.

The patient should hold the tube at the lips and the operator steady the bottle and compress the bulb. The spraying should be begun when the tube has entered to the mark (about 16 inches). It can be forced further in.

Einhorn advocates it to disinfect the mucous membrane of the stomach; for the application of astringents; and to relieve pain in gastralgia, as from ulcer, cicatrix, or cancer.

It is recommended in erosions of the stomach; in chronic gastritis, with marked production of mucus; in hypersecretion and hyperacidity, and in gastralgia.

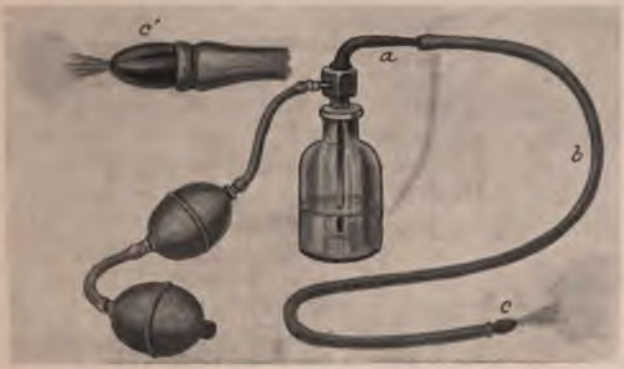

Fig. 93.—Einhorn's gastric spray.

Riegel holds that the necessary insufflation of air distends the stomach, and on this account it is objectionable. I have found the spray of value in gastralgia and in the treatment of erosions.

STOMACH POWDER-BLOWER

A dry method for spraying the stomach with insoluble substances (powders) has been devised by Einhorn.[1] His instrument consists of a flexible rubber tube about 28 inches long (Fig. 94), the distal end of which connects with an air suction-bulb.

The extremity of the tube is attached to a hard-rubber piece *c*, which is hollow and has lateral openings. It is provided with a screw thread. To this is attached a capsule with numerous side holes. Capsules of several sizes are furnished. A capsule is filled with powder and screwed on to the tip piece. A small spoon is employed to fill the capsule.

It is well to lubricate the latter with a thin layer of vaselin to prevent entrance of moisture. The tube is then dropped into warm

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[1] New York Medical Journal, April 1, 1899.

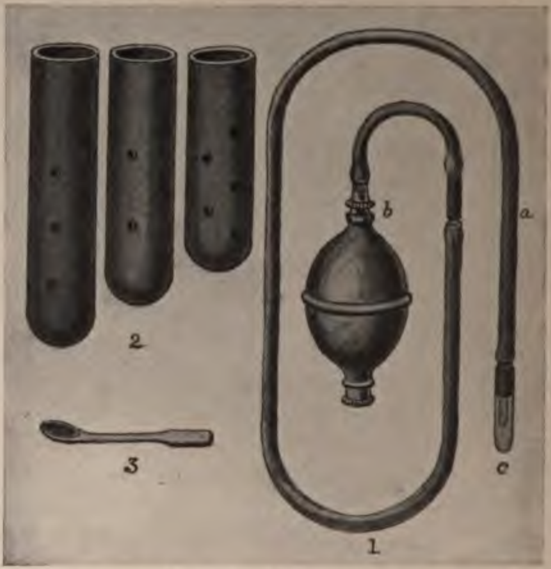

Fig. 94.—1. The stomach powder-blower: *a*, the tubing part; *b*, connection with the bulb; *c*, hard-rubber end with screw thread for capsule; 2, the capsule-shaped powder receptacles (natural size); 3, the small spoon for putting the powder into the capsule.

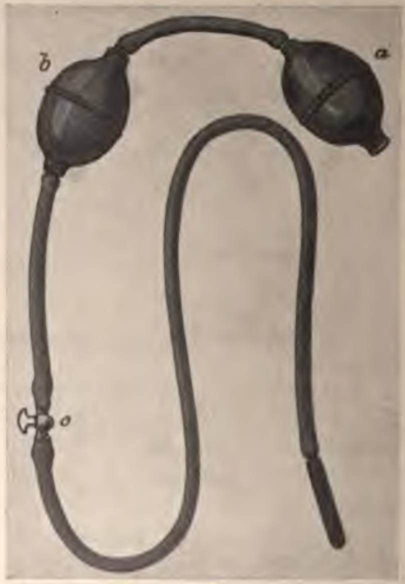

Fig. 95.—New model powder-blower.

water and inserted into the stomach in the usual manner. The bulb is quickly compressed several times and the air drives out the powder, opening up the vaselin layer over the holes.

It has been recommended for ulcer of the stomach, employing bismuth subnitrate; in gastralgia, orthoform; and in erosions, protargol or suprarenal powder.

I would not care to advocate the passage of the instrument in ulcer, but in the other conditions it is at times of service. Recently Einhorn has modified this instrument (Fig. 95). There is a double-bulb with a stop-cock. This last is closed. Bulb *a* is compressed several times, thus overdistending bulb *b*, which is without valves. The stop-cock is opened and a single blast of air distributes the powder; about 15 gr. (1.0) is the capacity of the capsule.

ELECTRICITY

From clinical experience it is found that the electric current exercises an influence on the secretory and motor functions of the stomach and also on its sensibility. Physiologic experiments and clinical experience do not always agree.

Meltzer, experimenting on animals, passed strong induced currents through the fundus of the organ and noted no contraction of the pylorus. The influence of the anesthetic or of morphin or similar drugs would influence the experiment.

Pepper demonstrated on a very thin patient that percutaneous electricity produced no peristaltic movements in the stomach. It is believed that it is through contraction of the abdominal muscles that this procedure influences the musculature of the stomach. The two methods for the application of the galvanic and faradic current are the percutaneous and the intraventricular. The *latter seems preferable from a therapeutic standpoint*, but the former is easier and is more readily submitted to by patients.

Percutaneous Method.—Von Ziemssen employs two large plate-electrodes, one anteriorly between the pylorus and fundus, and the other from the fundus to spinal column, with a separating space of only 2 cm. The electrodes should be moistened and sufficient current employed to cause strong contraction of the abdominal muscles. If smaller sponges are used, they can be moved about in these regions. The séance should last ten to fifteen minutes, and be carried out at first every other day, depending upon indications, which are the same as for the intragastric method.

Intragastric Method.—Kussmaul first suggested the internal application of electricity, and was the first to introduce the sound with a copper wire and olive point into the stomach. Bardet improved upon this, and employed an electrode which did not touch the stomach wall, the circuit being established by filling the organ with water.

Numerous intragastric electrodes have been devised, of which the most practical are Einhorn's, Lockwood's, and Bassler's.

Lockwood's Electrode.—This consists of a very small cable of conducting wire covered with rubber. The intragastric tip is olive pointed and protected from the gastric mucous membrane by rubber finistrated capsule (Fig. 96). The instrument in appearan is much like his gastrodiaphane. It is easy of introduction and such small caliber that it does not incommode the patient. A gla or two of water is administered on the empty stomach, and t instrument is passed along the roof of the mouth, like the stomac tube, about 18 inches or, preferably, until the resistance of t

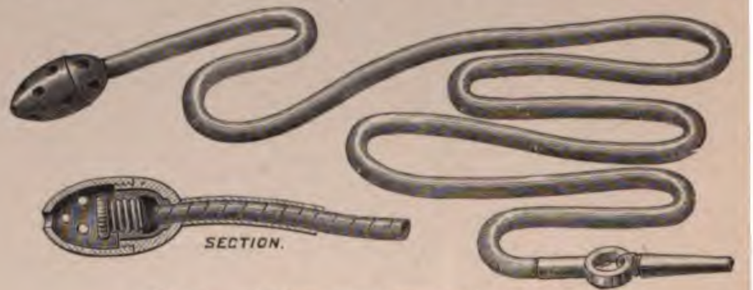

Fig. 96.—Lockwood's intragastric electrode.

stomach wall is encountered. It is then slightly withdrawn. T outer end of the instrument is furnished with a key which is insert into the negative pole of the battery. The sponge which is connect to the positive pole is applied over the stomach.

Einhorn's Deglutible Electrode.—The intragastric tip similar in construction to Lockwood's. The conducting wire is mu more flexible than Lockwood's and is covered with thin rubbe

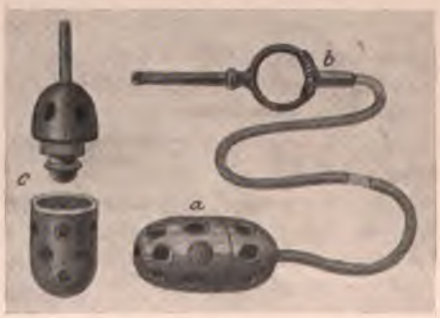

Fig. 97.—The deglutible stomach electrode.

It is not introduced by the operator, but swallowed by the patient being similar in principle to the stomach bucket (Fig. 97).

The key is connected to the cord of the negative pole of th battery. The patient drinks about a glass of water. The deglutibl electrode is placed on the root of the tongue and the patient i directed to swallow more water, which carries the instrument int the stomach. A mark can be placed on the cord, about 20 inche from the electrode, to determine that it has reached the bottom of the stomach. The sponge is attached to the positive pole. If there is resistance to the withdrawal of the instrument, the patient should swallow to relieve the spasm.

Bassler's Gastric Electrode.—This instrument is practically a combination of Figs. 96 and 97. It has the usual capsule with a cord-like conducting wire and also an introducer (Fig. 98).

The latter is withdrawn after the introduction of the electrode. After the application is completed the electrode is removed by the flexible conducting cord.

I have found Lockwood's electrode easy to introduce and unobjectionable to the patient. All are good instruments.

Gastrofaradization.—Duration ten to twelve minutes. The stomach electrode is attached to the negative pole. A plate electrode connected with the positive pole is placed in the epigastric region for four or five minutes, and later a sponge. The electrode is then moved from left to right several times in the gastric region; and later, if marked constipation, from the caput coli to the sigmoid along the colon, and also over the umbilical region. The application over the stomach occupies about two minutes. The electrode is then placed to the left of the seventh dorsal vertebra for one to two minutes, and then returned to the front for the balance of the time. The current should be strong enough to cause contraction of the muscles, *but not produce pain.* Application of the sponge to the intestines occupies several minutes, in addition to the ten minutes over the stomach. I sometimes reverse the poles during the application, by changing their external attachments to the battery.

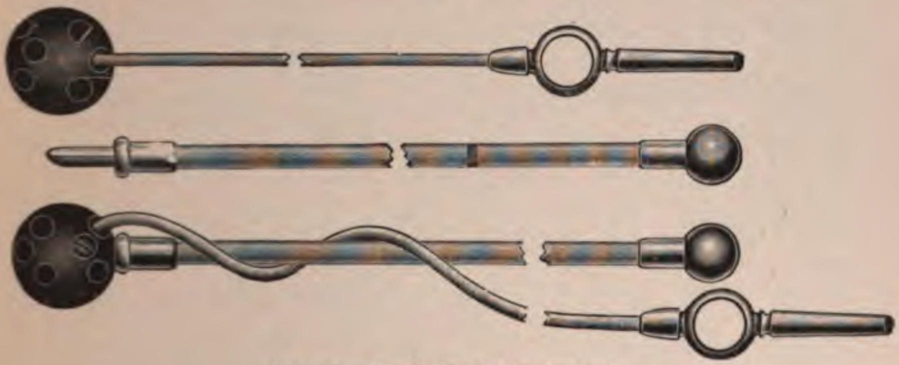

Fig. 98.—Bassler's gastric electrode.

Gastrogalvanization.—Duration eight to ten minutes. A small sponge electrode is placed on the epigastrium. The intragastric electrode is connected with the negative pole. The strength of the current should average 15 to 25 milliamperes. The full strength is not used at first. The application is two minutes to the epigastrium. It is then moved about over the gastric region for three to fo minutes; then for one to two minutes in the dorsal region as describe and the balance of the period in the gastric region.

There has been some dispute as to the effects of these curren and the indications for their use. In general we may say that dire faradization increases the gastric secretion, while galvanizati decreases it.

Faradization affects the musculature and galvanization t sensory field. The claims regarding increase in absorptive pow from the use of electricity seem hardly to be substantiated, as it w so small as to be within normal limits.

Therapeutics.—The percutaneous method has proved of servi in nervous anorexia and in motor insufficiency. The intragastı method is preferable when possible. It renders some patients mo nervous, and in such should be avoided.

Among the indications for gastrofaradization are: Aton ectasia, atony, relaxation of the cardia and pylorus, and diminish secretion.

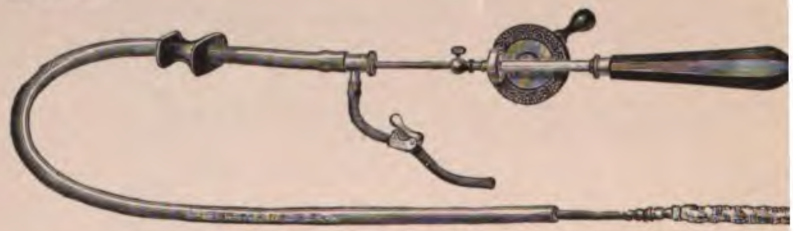

Fig. 99.—Türck's latest gyromele.

For gastrogalvanization they are: Gastralgias, especially nervous type, nervous anorexia, and hyperacidity.

Faradization, however, has proved of benefit in gastric neurose

Static Electricity.—This has been recommended especially the atonic types of ectasia, claim being made that it causes co traction of the organ. In neuroses it is sometimes of service.

High-frequency Current.—This method is scarcely applicab by the general practitioner, but I believe it of some benefit in gastr neuroses and in atony.

Türck's Gyromele.—Türck has introduced this instrument f the local treatment of the stomach and colon. It consists of a cab with a sponge attachment, which can be made to revolve within a outer stomach-tube. There is an arrangement so that medicate fluids can flow into the stomach or colon through the outer tub and also an attachment for a battery pole. He advocates its use f catarrhal gastritis to cleanse the mucous membrane. It can also b employed alone for internal massage of the stomach or combine with electricity. It is depicted in Fig. 99.

CHAPTER IX

MASSAGE—VIBRATORY MASSAGE—HYDROTHERAPY—COUNTERIRRITATION—ORTHOPEDIC APPLIANCES

MASSAGE

MASSAGE of the stomach is indicated in atony or in the atonic form of dilatation and to temporarily aid in the removal of gas. The intestines should also be manipulated in these conditions. It is of value in stimulating the abdominal muscles in gastroptosis. I will briefly refer to a few simple methods.

If massage is performed on the absolutely empty stomach it is contracted and cannot be palpated. It is preferable to perform it two or three hours after a meal. This aids in emptying the stomach. Earlier manipulation might cause vomiting.

Counterindications are ulcer, recent hemorrhage, and acute inflammation.

The patient should be in the dorsal position; lower limbs flexed. The left hand of the operator is placed on the right hypochondrium to exert counterpressure against the pyloric end. With the thumb and fingers extended the right hand performs stroking motions from left to right over the stomach. Then the stomach is kneaded. These procedures should alternate. This technic should be carried out daily for five to ten minutes. With dilatation or ptosis of the stomach the direction of the stroking must be adapted to the position of the organ in each case.

Tapping (tapotement) or rapid vibratory movements with the fingers can be employed. It is often well to rotate the patient to the right side during massage, so as to aid in emptying the atonic stomach.

VIBRATORY MASSAGE

Various vibrators, especially electric, many of which are quite expensive, have been devised for this purpose. There is an instrument run by carbonic acid gas pressure. These necessitate the electric current or carrying a large tank. There is a small portable vibrator, the Vedee, manipulated by hand (Fig. 100), which is cheap, simple of manipulation, and efficacious. The strength of the vibration is regulated by changing the position of the rotating disk. The instrument can be employed, with the addition of electricity, by attaching a sponge arranged for battery connection and employing the other pole with a sponge over the abdomen. Electric vibratory massage can thus be given over the stomach and intes-

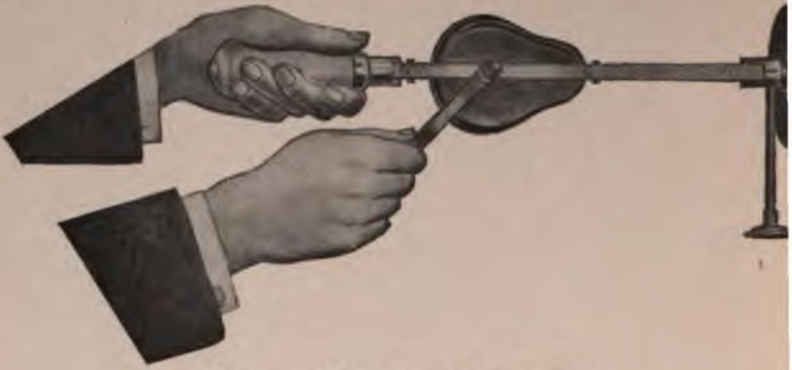

Fig. 100.—Vedee vibrator (new model).

tines (Fig. 101). In addition, one battery pole can be attached

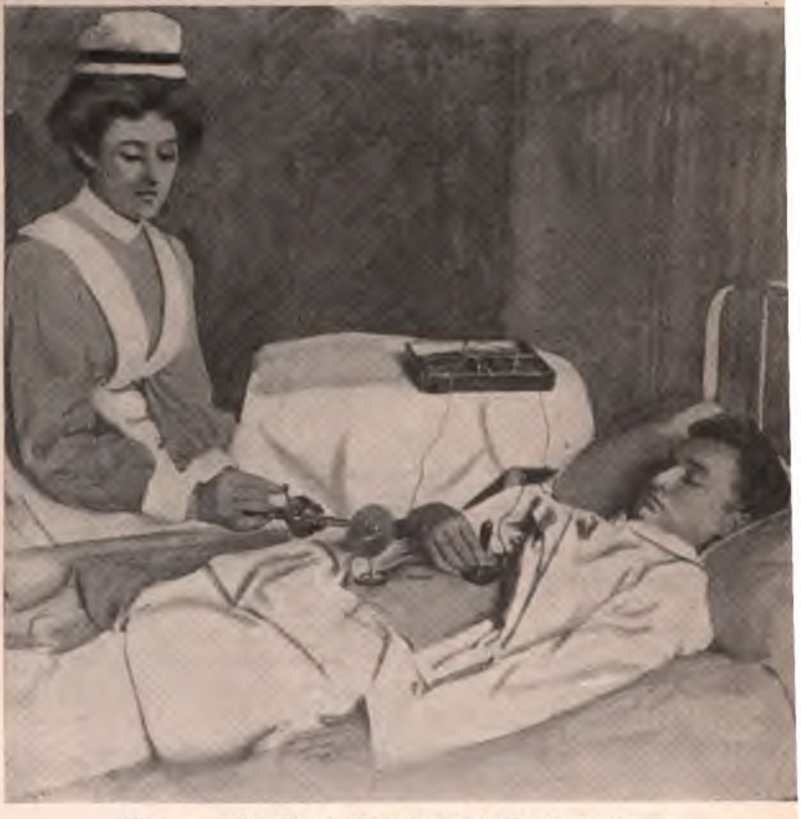

Fig. 101.—Combined electricity and vibratory massage.

the vibrator and the other to the intragastric electrode for treatment of atony of the stomach.

Vibratory massage should be given from left to right over the stomach for about three minutes, then two minutes to the left of the seventh dorsal vertebra, and three minutes more over the stomach. This should be performed daily.

It is generally advisable to vibrate the intestines, following the course of the colon, especially over the sigmoid, and also over the small intestine. This last can be done before breakfast to stimulate the bowels. Two or three hours later the stomach should be vibrated. The patient's family can be taught to use this instrument.

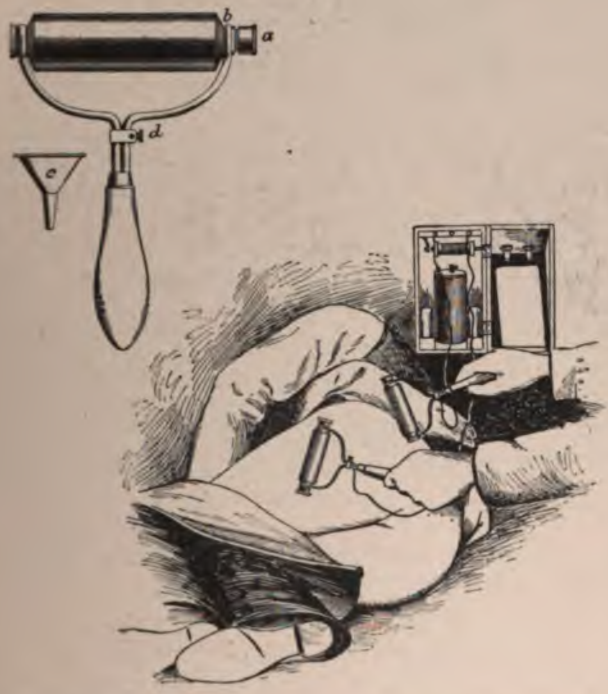

Fig. 102.—Massage roller with demonstration of alternating hot and cold electric massage: *a*, Screw cap; *b*, filling tube; *c*, funnel for filling; *d*, attachment for battery pole.

Bassler has recently devised an excellent vibrator, but it requires the street current.

Massage Roller.—Various instruments have been devised, but the following is simple. It consists of a revolving cylinder, which can be filled with hot or cold water, and which is furnished with a battery attachment. Heat or cold, or with an instrument attached to each battery pole, alternating heat and cold, combined with electricity, can be applied (Fig. 102). This method can be employe for abdominal massage in some cases of chronic constipation.

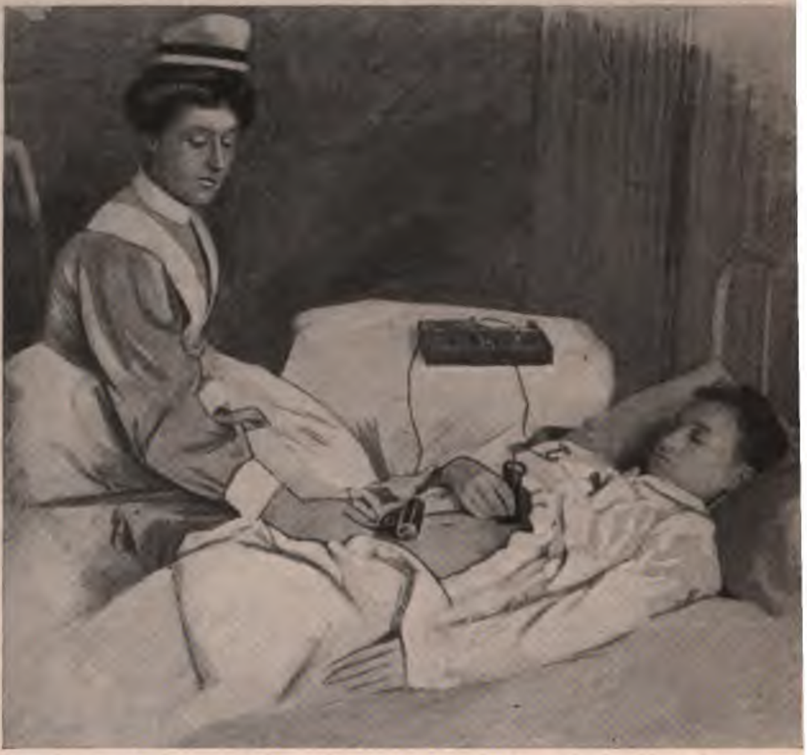

Fig. 103.—Combined application of electricity and roller massage with heat.

I have found the method next depicted of some value in stimulating atonic conditions of the gastro-intestinal tract (Fig. 103).

LOCAL HYDROTHERAPY

Cold.—For hemorrhage or acute inflammation, the ice-bag is preferable to the Leiter coil. At times it relieves ulcer pain more than does heat.

Priessnitz's Compress.—A towel folded several times is dipped in cold or warm water, then wrung out, and placed over the stomach. Oiled silk or gutta-percha is placed over it, and a flannel binder applied to keep it in place. A temperature of 50° to 75° F. or warmer if desired can be employed.

This method is of value in nearly all painful diseases of the stomach. The compress can be changed two or three times a day. Some patients do better with the cold, others with the warm compress.

Hot Applications.—*Moist Heat.*—In cardialgia, ulcer, vomiting, etc., hot moist applications are of value. Poultices can be made of linseed, flaxseed or bran, boiled in water, or of bread and milk. I have seen hot meal or hot mashed potatoes used in country practice.

The poultice is wrapped in gauze or cheese-cloth and applied as hot as the patient can bear it. Fresh hot poultices are continually applied. There is an apparatus which can be boiled in water, wrapped in a cloth, and then applied over the cataplasm. It will keep it constantly hot.

A felt sponge dipped in boiling water, wrung out, and covered with oiled silk can be employed.

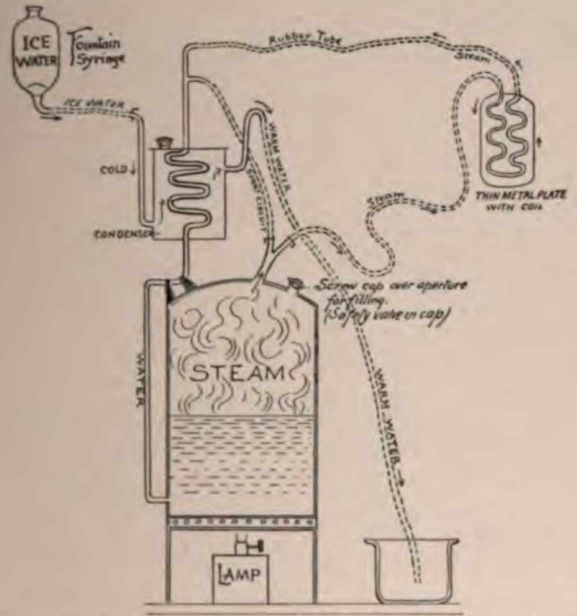

Fig. 104.—Continuous steam coil for the application of heat.

Dry Heat.—The hot-water bag, hot cloths, a light tin pieplate heated in the oven and covered with flannel, the Japanese hot box containing burning punk, are all useful. A continuous hot-water coil has been devised.

In the illustration (Fig. 104) is shown a continuous steam coil[1] of my own. The steam passes through a metal coil plate and is recondensed. Only a small quantity of water is necessary in the boiler. The temperature can be regulated by the coverings of the plate and by the stop-cocks on the Y-branch at the top of the boiler, thus allowing less steam to enter the coil. The coil can be placed over a moist poultice to preserve its heat. In general, moist compresses are preferable.

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[1] Enteroclysis, Hypodermoclysis, and Infusion, 1900.

The fan douche and the Scotch douche, played over the stomach region, alternating cold 55° F. and warm 95° F., for about three minutes, may have a tonic effect. Packs, rubs, baths, and the carbonated bath are at times employed for the general tonic effect.

COUNTERIRRITATION

Mustard and flour poultice (equal parts); or black or red pepper, ʒj (4.0) to the pint (500 cc.) of boiling water, flannel to be wet therein, wrung out, and applied with an oil silk cover or a turpentine stupe, prepared by the same method, are of service.

ORTHOPEDIC METHODS

Mechanical support of the stomach and intestines is of great service. Many nervous symptoms referred to the gastro-intestinal tract can be imputed to ptosis of the viscera and relaxation of the abdominal walls. In simple atony of this tract the proper support frequently aids the bowel action.

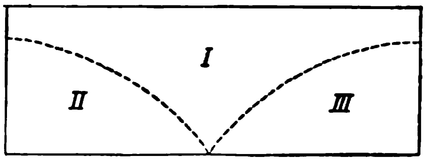

Fig. 105. Pattern for cutting the Rose plaster abdominal binder: Dotted lines for section.

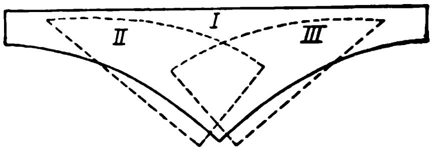

Fig. 106 - Pattern for cutting the Rose plaster abdominal binder: Plaster after section.

The indications for mechanical support are as follows:

Ptosis of any or all of the viscera from any cause; atony of the stomach; atonic dilatation of the stomach; stenotic ectasia as a temporary measure; atony of the intestines; pains from intra-abdominal adhesions dragging on the viscera; hernia of the abdominal wall; after laparotomy as a temporary support; post-partum to prevent ptosis (Landau's disease), and also to enable the patient to sit up in bed earlier and so drain the uterus. This last was suggested by me to Douglas H. Stewart, who has reported successful results. Cases of constipation in which atony is a factor. Pelvic disturbances which are associated with splanchnoptosis and result from general prolapse. Mucous colic in which ptosis is a factor. Pendulous abdomen, vomiting of pertussis (T. W. Kilmer), seasickness, and nervous vomiting.

There are three methods: The use of adhesive plaster, the most scientific; elastic bandages, and a special corset.

To A. Rose we must credit the best method of adhesive strapping. The author first suggested the use of zinc oxid on moleskin plaster, from experiments finding it most suitable, and this was adopted by the originator of the method. For a full description I would refer to our work on this subject.[1]

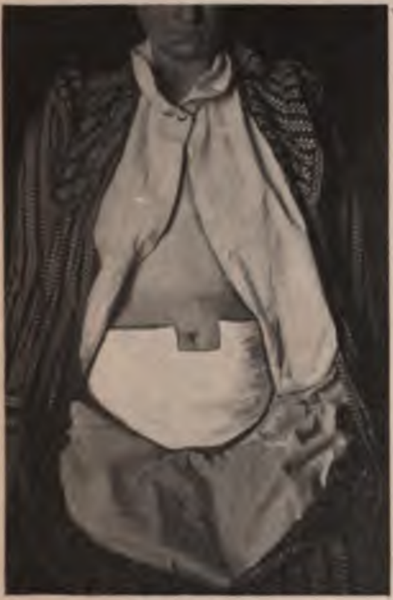

Fig. 107.—Rose's belt (under plaster): Step one.

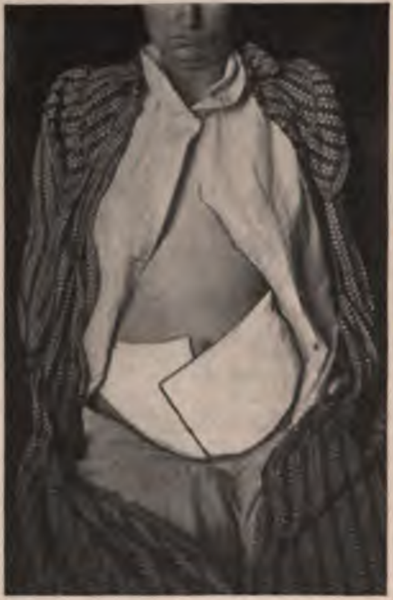

Fig. 108.—Rose's belt: Step two, left wing.

The method is as follows: Adhesive plaster, zinc oxid on soft moleskin (Johnson and Johnson), preferably 7 inches wide, though 6 inches can be employed. A yard in length will suffice in most cases. The circumference of each patient should be measured, and the plaster should be long enough to encircle the waist. The plaster is folded over so that the free ends are in line and a curved line drawn in pencil from the lower margin of the point where it folds to the free margin, to about 1 inch below the upper border. The plaster is cut along this line, giving three pieces; or the plaster is stretched out and the dotted lines marked, as in Fig. 105, and cut along these lines, giving three pieces, *I* and the two lateral pieces, *II*, *III*, as in Fig. 106.

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[1] Rose and Kemp, Atonia Gastrica.

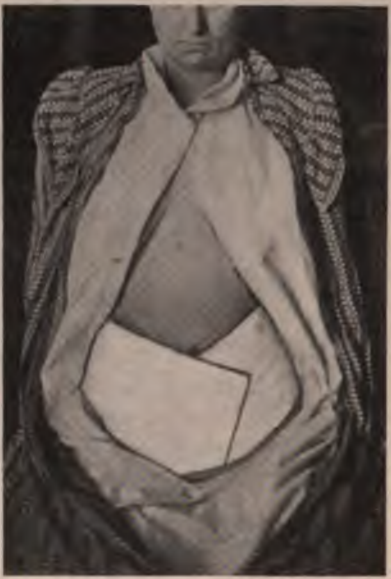

Fig. 109.—Rose's belt: Complete.

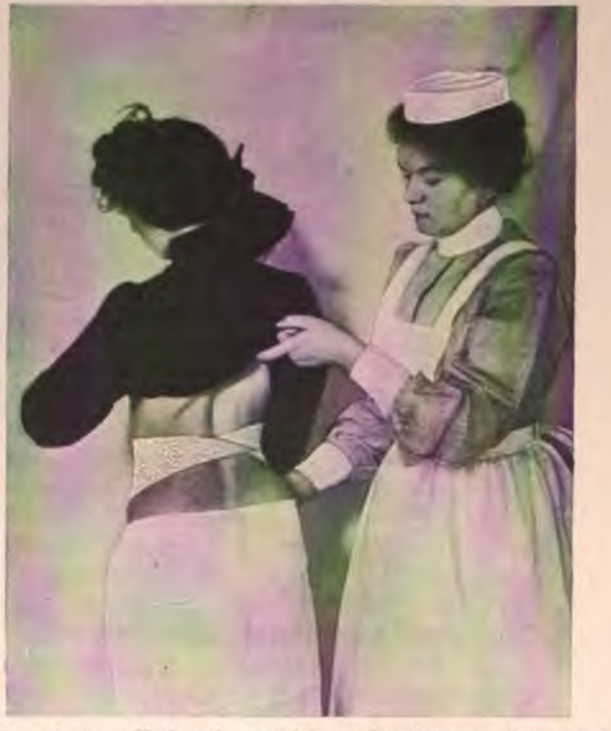

Fig. 110.—Dorsal view: Under plaster with overlapping ends (Rose and Ke

I is applied to the abdomen, and the lateral pieces, *II, III*, overlap in front and are applied to the under plaster. These serve to draw up the abdomen.

To avoid irritation of the umbilicus, I cut a **V** out of the upper border of the under plaster or invert a small portion of it. The sharp angle below should be cut off to avoid interference with the pubic hair. The curved portions of the lateral wings should look upward and somewhat inward and adhere to the lower ribs. The sharp angles of the lateral wings at the symphysis may also be cut off to avoid the hair.

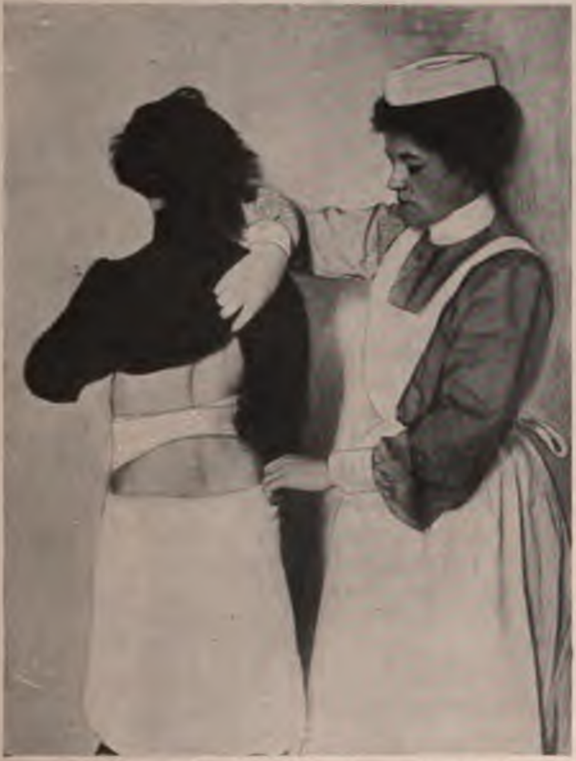

Fig. 111.—Dorsal view: Plaster dressing complete (Rose and Kemp).

Hair, if present on the abdomen, is shaved and the surface cleaned with ether or chloroform.

The plaster is applied with the patient in the dorsal position, and preferably with hips slightly elevated.

In the illustrations (Figs. 107–109) are shown the three stages of application of the belt, the under plaster, one wing applied, and the complete contrivance. In Figs. 110 and 111 are shown the rear view during application.

The plaster should remain on for four to six weeks, depending on the season of the year, irritation (which is rare), or its loosening.

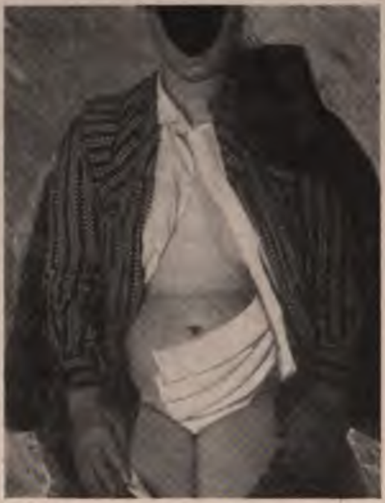

Fig. 112.—Application of narrow strips of adhesive plaster: First step.

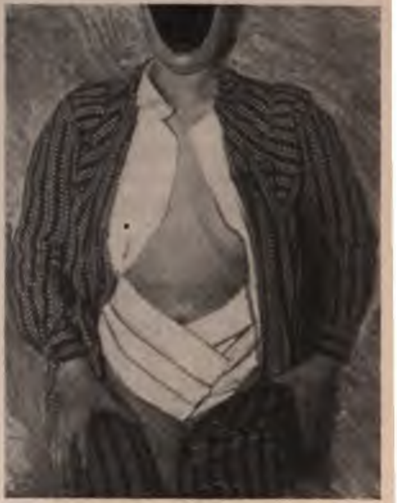

Fig. 113.—Application of plaster: Second step.

It should then be removed, a full bath given, talcum dusted on, and twenty-four hours later a new belt applied.

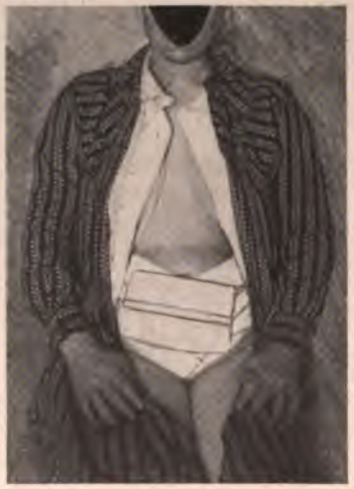

Fig. 114.—Application completed.

One patient sent me by Wm. H. Thomson, a severe case of splanchnoptosis, wore the belt fourteen months, gained 40 lbs. in weight, and was completely cured.

The device gives brilliant results. The method of support by a pad for the special organ is unscientific.

Only on occasions when the material for Rose's belt was not at hand have I applied a method with narrow strips of plaster, as depicted in Figs. 112–114. They overlap at the linea alba in front and at the spine behind. As the final procedure, two transverse strips are applied in front.

Rosewater Adhesive Belt.—A strip of zinc oxid plaster of sufficient length and two to three inches wide is fastened to the abdomen just above the pubes. This is drawn upon upward and fastened above to the lower end of the sternum. Diagonal strips crossing the lower end of the vertical strip, overlapping behind at the spine, are then applied (Figs. 115 and 116).

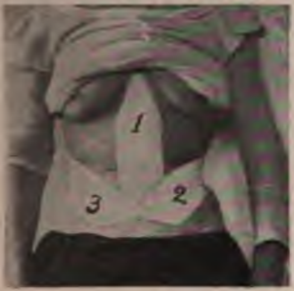

Fig. 115.—Front view: Step one.

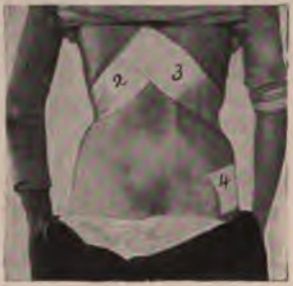

Fig. 116.—Rear view.

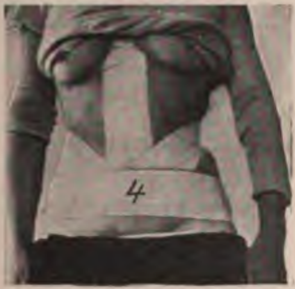

Fig. 117.—Front view. Belt complete.

Figs. 115–117.—Rosewater adhesive plaster belt (Rose and Kemp).

A horizontal strip is fastened to one hip and stretched across the pubes to the other hip, overlapping the ends of the other plaster and acting as an additional girdle (Fig. 117).

Plaster strapping affords continuous support during treatment, which other methods do not. Numerous modifications have been devised, but these are the most practical.

Kilmer's Abdominal Belt.—An ingenious belt was devised some years ago by T. W. Kilmer for the relief of vomiting in pertussis. The original instrument consisted of a stockinet band applied as

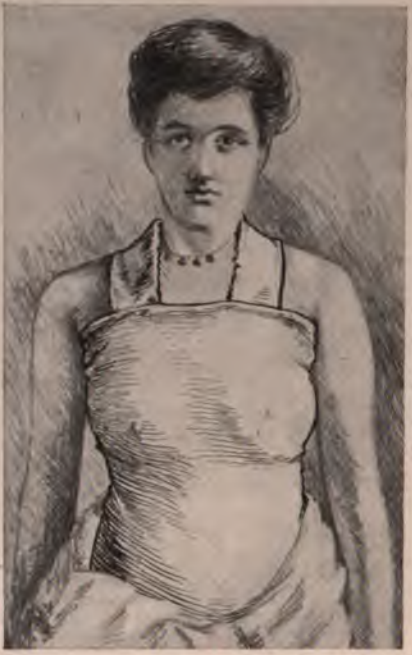

Fig. 118.—Kilmer's belt stockinet band: Step one.

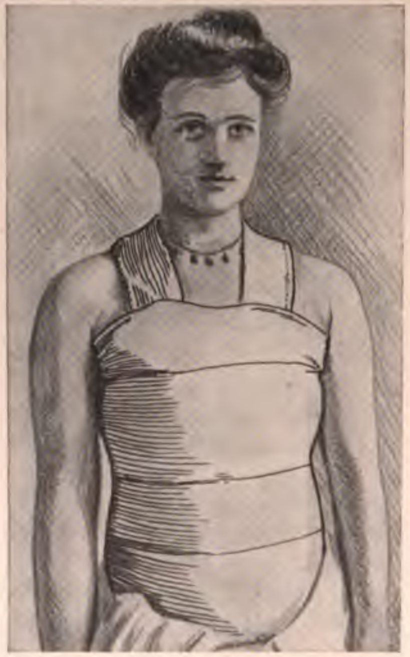

Fig. 119.—Kilmer's belt (complete): Elastic webbing, front.

in Fig. 118. Around this was wound a strip of silk elastic webbing, which could be pinned or sewed on, as in Figs. 119 and 120.

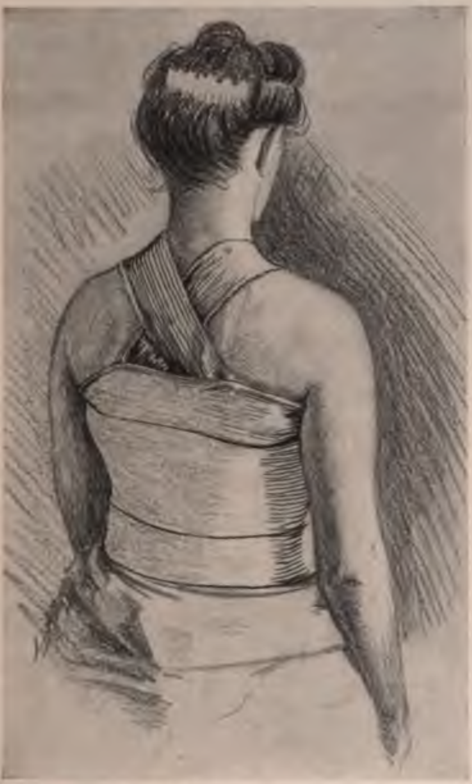

Fig. 120.—Kilmer's belt (complete): Elastic webbing, rear.

The apparatus extends from just above the hips and symphysis well up on the thorax. The relief of vomiting and cough was quite remarkable in pertussis.

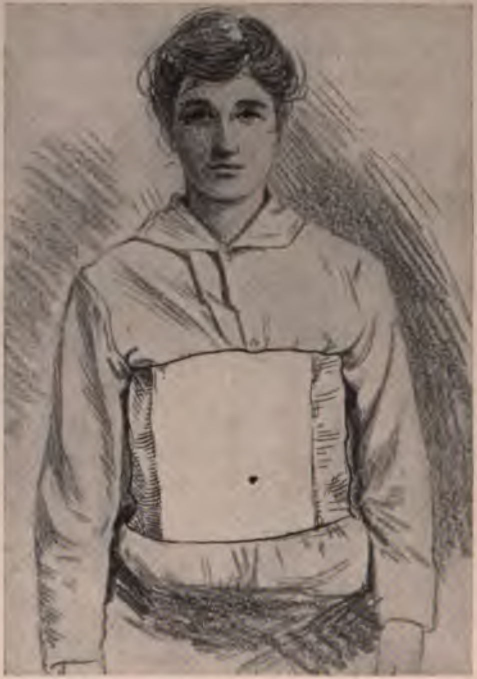

Fig. 121.—Improved belt (front).

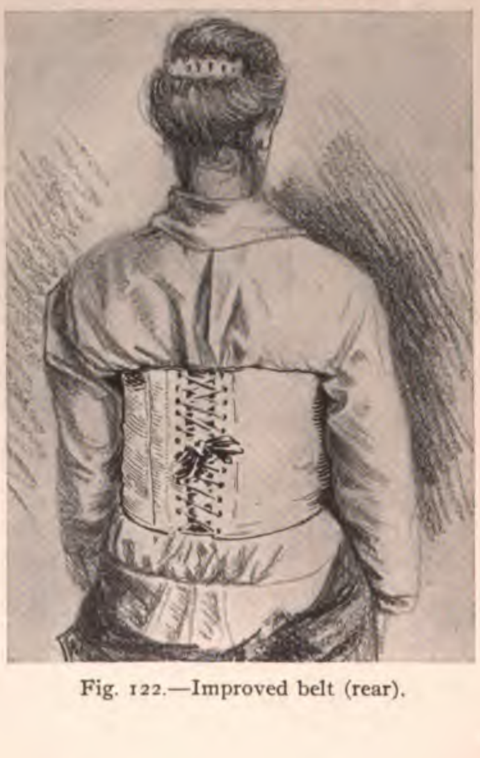

Fig. 122.—Improved belt (rear).

Recently Kilmer reports a simplified belt[1] made of linen, with strips of elastic webbing inserted on either side. It laces up the back (Figs. 121 and 122).

The belt should measure slightly less (2 to 3 inches) than the circumference at the navel. The degree of constriction should be determined in every case. The instrument is valuable for the prevention of seasickness, for nervous vomiting, and as an abdominal support. The cost is slight.

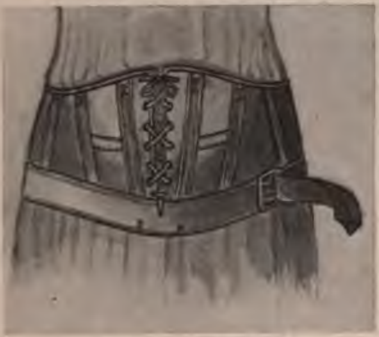

Fig. 123.—Teufel's abdominal supporter.

Silk Elastic Belts.—Various types of silk elastic belts are employed. Figs. 123–125 show useful instruments. Storm's abdominal supporter is also excellent.

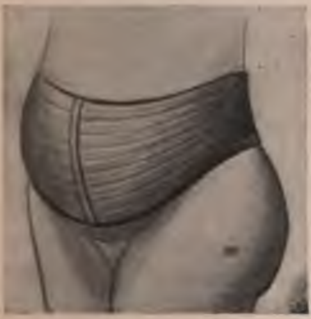

Fig. 124.—Abdominal supporter.

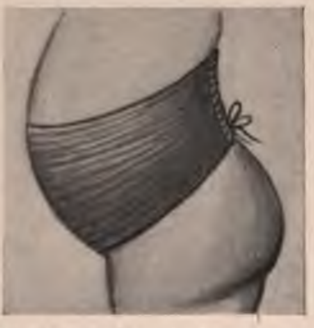

Fig. 125.—Abdominal supporter.

Special Corsets.—The most valuable support of this type is that of E. Gallant, which is depicted under Gastroptosis. The La Grecque surgical corset is also useful, and is illustrated in the same chapter (Fig. 161).

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[1] Archives of Pediatrics, February, 1907

CHAPTER X

CATARRH OF THE STOMACH

ACUTE AND CHRONIC GASTRITIS

ACUTE GASTRITIS

ACUTE gastritis may be defined as an acute inflammation of the gastric mucous membrane with resulting disturbances of digestion. It is of different degrees of severity, being limited to the superficial layer of the mucous membrane, or it may extend to the glandular parenchyma or involve the interstitial tissues.

It is subdivided into simple acute gastritis, toxic gastritis, and phlegmonous gastritis.

SIMPLE ACUTE GASTRITIS

(*Synonyms.*—Acute Gastric Catarrh; Acute Gastric Dyspepsia.)

Etiology.—Simple acute gastric catarrh is one of the most frequent diseases met with by physicians. It occurs in all classes of society and at all ages. It may be primary or secondary to another disease. One of its frequent causes is some irritant, mechanic, chemic, or thermal; thus errors in diet, or too large quantity of food that has been imperfectly masticated, or rapidly bolted; too hot or too cold food or drink; too highly spiced or fermented foods; rancid butter; unripe or spoiled fruit; spoiled food or drink, or overindulgence in alcohol.

Fermentation or putrefaction are most apt to occur in food during the summer, and these factors probably account for the epidemics occurring at that season, though infection has been suggested. Such cases generally occur as gastro-enteritis.

Bacterial infection of food may be a cause, as the colon bacillus in milk, or meat, or sausage poisoning.

Primary mycosis of the stomach, the favus fungus; schizomycetes; parasites, as the larvæ of flies; ascarides, oxyuris and tænia, by entering the stomach, and abdominal burns are rare causes. Acute catarrhal or suppurative conditions of the nose and throat may produce acute gastritis from the ingestion of discharges. I have recently seen one such case and the patient suffering from acute gastritis, cured by treatment of the source.

Some persons have a predisposition to a "weak stomach," and this condition seems almost to be hereditary. Others have been trained to such a simple diet, as the children of dyspeptics, that the stomach cannot perform its normal amount of work and readily becomes irritated. In old persons, invalids, or anemic women the organ is readily affected.

Acute gastritis may be secondary to the acute infectious diseases, such as measles, typhoid, variola, pnuemonia, etc., or as a sequel of acute nephritis. I have seen an attack follow prolonged anesthesia.

A diphtheritic or membranous gastritis has been met with in diphtheria, or as a secondary process in typhoid, typhus, pneumonia, etc. It cannot be diagnosed unless the membranes are vomited.

Morbid Anatomy.—Acute gastritis is characterized by an acute inflammation of the superficial layers of the mucosa, with an increased secretion of mucus and a desquamation of the epithelial cells.

The mucous membrane is reddened and swollen, less gastric juice is secreted, and mucus covers the surface. The swelling is diffuse or in circumscribed areas. There are sometimes slight hemorrhages and small erosions or sacculations of the mucous membrane. The submucosa may be edematous. The pyloric end is more frequently affected. Gastric secretion is weakly acid, neutral, or even alkaline and diminished in quantity. Beaumont, from his observations on St. Martin, has given an excellent description.

Microscopic.—The superficial epithelial layer is partially loosened or in a condition of cloudy swelling. The parietal and principal cells cannot be distinguished apart; they are granular and in a condition of cloudy swelling and fatty degeneration, and are shrunken. The capillaries are dilated and round cells are found in the interglandular tissue, between the epithelial cells and on the surface. Karyokinesis may be present.

Symptoms.—These vary according to the severity of the attack. There is loss of appetite, discomfort, fulness or pressure in the region of the stomach, belching of gas, which may taste sour, and occasional nausea. In some cases there are no rise of temperature and no vomiting, and the symptoms pass off in a day or two; the bowels are costive or diarrhea is present.

In more severe cases there are pains in the gastric region, headache, nausea, vomiting (prolonged and excessive), first of food, then chiefly of mucus, at times streaked with blood, and frequently bilious vomiting. There may be considerable prostration. Often there is an acid taste in the mouth. There is generally a temperature, sometimes rising to 102° to 104° F., and at times chills, and the tongue is usually coated and swollen. The pulse is frequently rapid and feeble.

In the cases due to ingestion of spoiled food, etc., auto-intoxication undoubtedly results and aggravates the symptoms.

Vomiting usually follows the introduction of the irritant, but is sometimes delayed for some hours, and food is found that was ingested twelve or fifteen hours before, a condition of acute motor insufficiency. Constipation or diarrhea is present. The early vomitus often has a disagreeable odor and contains food remnant The reaction is slightly acid and free hydrochloric acid is ofte absent. Lactic and other organic acids are at times present. Occasionally a duodenitis with jaundice is associated. Herpes labialis i quite frequent.

Physical Examination.—The region of the stomach is usuall distended and sensitive, and may be tender on pressure.

Urine is scanty, dark in color, of high specific gravity, urate marked, and occasionally indican and albumin.

Duration.—This is usually short, from two to three days, thoug at times prolonged to a week.

Diagnosis.—Some of the infectious diseases, notably scarlatina begin like the febrile form of acute gastritis, and one should alway be on the watch for such an occurrence.

In rare cases the symptoms are intensely severe, headache an even delirium being so marked as to have been mistaken for meningitis. In the latter case, Kernig's sign can be elicited and lumba puncture is an aid to diagnosis.

In biliary colic, with acute vomiting, the pain radiates to th right side or right shoulder, and pain over the gall-bladder is present

In cholecystitis, with or without calculi, with little or no pai and no jaundice,[1] but vomiting, the diagnosis is more difficult. Th previous history, tenderness over the gall-bladder, and the presenc of leukocytosis, especially the increase in the polynuclears, ar significant. Hyperchlorhydria is frequently associated.

With peritonitis, we have muscular rigidity, leukocytosis, increased polynuclears, marked abdominal tenderness, and distention.

With nervous gastralgia, the material vomited is very acid (hyperchlorhydria), no mucus, and the nervous history.

With typhoid fever, we have the gradual rise of temperature, increasing daily, the splenic enlargement, frequently the eruption, often bronchitis, Ehrlich's diazo and the Widal reactions; while, with acute gastritis, the rise of temperature is sudden and the fall equally sudden, and there is the absence of splenic enlargement and other symptoms.

When jaundice is associated with acute gastritis, the duodenum has evidently become involved. The gastric crises of locomotor ataxia have been mistaken for acute gastritis, but the absence of knee-jerks, the Romberg symptom, and Argyll-Robertson pupil are diagnostic of tabes.

Prognosis.—This is favorable, except in very old people and invalids.

Treatment.—*Prophylaxis.*—In patients subject to attacks of acute gastritis, excess in eating, rich food, lobster, food and drink that are too hot or cold, or any articles for which they have an idiosyncrasy should be forbidden. Candy and cake should not be allowed. Unripe and dirty fruit should be avoided. With infants, care should be taken as to the storage of milk and its preparation.

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[1] The presence of Head's gall-bladder zone of cutaneous algesia aids diagnosis.

If the acute gastritis be due to ingestion of improper food, there are two principles to follow: Clear out the gastro-intestinal tract and give rest to the stomach. If there are other factors, the first consideration does not apply. In all cases *rest in bed should be enjoined.*

In the *mild cases*, with *nausea* but *no vomiting*, castor oil, ℥j to iss (32.0–48.0); or, if there is doubt of this being retained, then calomel, gr. 2 to 5 (0.125–0.3), or blue mass, gr. 5 (0.3), followed in twelve hours by a saline, such as a wineglass of apenta, or citrate of magnesia (wineglass), or magnesia sulph., ʒj to ij (4.0–8.0), should be administered.

If much nausea, calomel, gr. $\frac{1}{10}$ (0.006), with sodium bicarb., gr. $\frac{1}{2}$ (0.0325), every hour for eight or ten doses, followed by a saline.

Children should receive proportionately small doses of cathartics.

For eructation of acid fluid (pyrosis), bismuth subnit., gr. 3 (0.2), with sodium bicarb., gr. 3 (0.2), every hour or two; or magnesia usta, ℥ss (16.0), with sodium bicarb., ℥ss (16.0), as much as covers the point of a knife, every three hours.

For nausea or vomiting, oxalate of cerium, gr. 1 (0.065), every hour for several doses, or bismuth subnit., gr. 2 to 4 (0.125–0.25), alone, or with sodium bicarb., same dose; or gelatin (1 per cent. solution), dose ʒj (4.0), every hour, given cold; or milk, ℥viij (250.0 cc.), with oxalate cerium, gr. 10 (0.6), and sodium bicarb., gr. 10 (0.6), packed in ice; dose, ʒj (4.0) every hour; or Fowler's solution of arsenic, ♏j (0.06), every hour for four doses, are of value.

Teaspoonful doses of hot water or, occasionally, a piece of cracked ice; or white of raw egg beaten up and given cold in 1-dram doses (4.0), every half-hour to an hour, are useful.

Cocain, carbolic acid, or creosote I strongly deprecate. Heat should be applied.

Application of Heat.—A hot-water bag, one-third full to avoid weight; a Japanese hot box, a light hot salt-bag, a thin tin pieplate heated in the oven and covered with flannel; or moist heat by means of a flaxseed poultice, hot mashed potato or bread poultice in country practice; mustard and flour poultice (equal parts); or black or red pepper poultice—ʒj (4.0) to Oj (500 cc.) of boiling water—and wring out flannel in the same and apply, covering with oiled silk.

If the discomfort is marked, then give warm salt water, ʒss (2.0) of salt to ℥viij (250 cc.) of water, and tickle the fauces to promote vomiting, or lavage.

Small doses of hot water may be given to relieve thirst and *food should be avoided for twenty-four hours.*

If small quantities of nourishment be given, I have found ℥j (32.0) doses every hour of a 5 per cent. gelatin solution (in a glass packed in ice) of special value, or small quantities of milk and lime water (equal parts), or peptonized milk or kumyss.

In *severer cases*, the stomach should be emptied, preferably b lavage, ℥j to ij (32.0–64.0) of Phillips' milk of magnesia to quarts (liters) of warm water being excellent for this purpose Plain warm water or normal salt solution may be employed.

I frequently administer calomel, gr. 3 to 5 (0.2–0.3), and sodiu bicarb., gr. 5 (0.3), in a little water, through the stomach-tube afte lavage before withdrawal of the tube. This is generally retained with resulting thorough clearing of the bowels. Saline solutio should not be employed for lavage if calomel is thus given.

Later an enema of saturated solution—℥ij to iij (64.0–96.0)– of magnesium sulphate, or a recurrent enteroclysis with norma saline solution at 110° F. should be administered (several quarts– liters), or a soapsuds enema, 1 quart (liter), containing olive oil ℥vj (200 cc.), may be substituted. Even if the calomel be omitted it is of great importance to move the bowels. If the case is not du to ingestion of irritating food, then bismuth subnitrate, oxalate o cerium, a few doses of gr. $\frac{1}{10}$ (0.006) of calomel every hour, sodiu bicarb., etc., may be tried for a brief period; and if these fail, lavag should be performed. Gelatin solution or white of egg often are o value in such cases.

With bilious vomiting, lavage should be employed at once, a there is practically reversed peristalsis with an open pylorus, an the continuous accumulation of bile in the inflamed stomach keep up the vomiting. Enteroclysis should be used after lavage (withi one hour) to promote normal peristalsis.

It may be necessary to wash the stomach several times, but it i the best method to check vomiting.

In the severe cases, due to ingestion of improper food, it is m opinion that calomel should be given directly after lavage, eve though some of it be vomited, as auto-intoxication is a factor.

In addition to the milk of magnesia, if the vomitus is very foul, I add gr. 10 (0.6) resorcin to the fluid for lavage and wash with the patient both in the erect and lying-down position. I do not care for apormorphin, gr. $\frac{1}{10}$ (0.006), ipecac, or tartar emetic to empty the stomach.

If there is considerable prostration, enemata of hot normal salt solution (115° F.) and strychnin by hypodermic—gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.002)—may be necessary.

In rare cases, when there has been much vomiting and the patient is exhausted, codein, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), by hypodermic, or the same amount of morphin may be used. I sometimes employ a single dose after lavage to quiet the patient. A suppository— gr. 1 (0.064) opium, or gr. $\frac{1}{4}$ (0.016) morphin with gr. $\frac{1}{3}$ (0.021) extract belladonna—may be substituted.

Diet.—Entire abstinence from food during the first twenty-four hours or longer is preferable; nutritive enemata may be given and injections (rectal) of hot saline solution to relieve thirst.[1]

For the latter, ʒj (4.0) doses of hot water by mouth, or of a cold 1 to 2 per cent. gelatin solution are preferable to cracked ice. The gelatin seems to have an excellent effect.

Later small doses of cold gelatin (5 per cent. solution) or milk and lime-water (equal parts) can be given, or milk, ℥viij (250 cc.), with sodium bicarb., gr. 10 (0.6), and oxalate cerium, gr. 10 (0.6), in ℥ss to j (16.0–32.0) doses, given cold, every two or three hours.

White of egg beaten up and given cold; barley-water alone or with milk (equal parts); rice gruel, very thin, made from rice flour (Park and Tilford) with milk may be added.

Later add eggs (soft boiled), scraped raw beef, pigeon (boiled), calves' brains, zwieback, broths, soups, bouillon, boiled chicken, and gradually increase to full diet.

If after the acute attack has subsided the patient suffer from a feeling of pressure and discomfort, dilute hydrochloric acid may be given to aid digestion.

℞. Acid. hydrochlor. dilut. ʒiiss (10.0);
Aq. destil. . . . . q. s. ad. ℥ij (60.0).
Sig.—ʒj to ij (4.0–8.0), in water, t. i. d.

The same combined with small doses of tincture of nux vomica, ♏v (0.3) in each dose; or with compound tincture cinchona, ♏x (0.6) in each dose may be given.

These remedies should be administered one-half hour after meals or the same time before.

TOXIC GASTRITIS

Etiology.—This most intense form of inflammation of the stomach is caused by the swallowing of concentrated mineral acids or strong alkalis, or by poisons, such as phosphorus or arsenic. Among such are nitric, sulphuric, hydrochloric, oxalic, and carbolic acids; the caustic alkalis, as caustic potash, caustic soda, soap lees, and strong ammonia; alcohol, phosphorus, arsenic, potassium cyanid, corrosive sublimate, and potassium chlorate. The effects are more severe on the empty stomach.

Anatomy.—The acids and alkalis destroy the parts they come in contact with, causing various degrees of sloughing of the mucous membrane. They may penetrate the submucosa or the entire stomach wall and produce perforative peritonitis.

Alcohol, phosphorus, or arsenic cause an acute inflammation of severe type, the mucous membrane becoming swollen and superficially necrotic, with hemorrhagic spots in the submucosa, and there is fatty degeneration of the epithelia of the glandular tubuli.

Symptoms.—There are intense pain in the gastric region, violent and burning in character, and increased on pressure; and frequently pain in the pharynx and esophagus (along the sternum). There are salivation, difficulty in swallowing, and usually vomiting, constant and repeated, which fails to relieve the pain. This is generally immediate, though not always so. There may be food remnants in the vomit, mucus, streaks of blood, and even shreds of mucous membrane. The abdomen is tender, at times distended, though occasionally contracted. Symptoms of collapse often appear, the face pale and anxious, the skin pale and extremely cold, pulse rapid and feeble, respiration rapid and shallow. There are restlessness and sometimes convulsions.

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[1] Proctoclysis is also excellent.

Albumin and blood often are present in the urine and petechiæ under the skin. Peritonitis, shock, or respiratory or cardiac failure may cause the fatal issue.

Some cases are more protracted. There may be jaundice or hematuria, or intestinal ulceration, or degeneration of the liver or kidneys. Phosphorus-poisoning may produce hemorrhagic jaundice and symptoms simulating acute yellow atrophy of the liver.

Stricture of the esophagus, pylorus, or cardiac orifice of the stomach can result from the damaged mucous membrane, with corresponding symptoms, such as dilatation of the stomach, etc. In rare cases, atrophy of the mucous membrane (achylia gastrica) follows, or hour-glass stomach, or perigastric adhesions.

Diagnosis is usually easy. The sudden appearance of violent gastric symptoms in a perfectly healthy subject should excite suspicion.

Inspection of the lips, mouth, and tongue will show the effect of corrosive poison, if such have been taken, and examination of the vomitus and odor of the breath may afford information. Inspection, if possible, of the receptacle from which the substance was taken, and examination of the latter, if any remain, are important. The patient often gives a clear history.

Prognosis.—It is best to give a guarded prognosis even in apparently favorable cases.

Treatment.—There are certain cardinal rules to follow in the treatment of toxic gastritis: first, administer fluid to dilute the poison, and at the same time give an antidote; empty the stomach as rapidly as possible, preferably by lavage; administer demulcents; stimulate the patient and give a cathartic to clear the poison from the bowels.

Though some advise against lavage in poisoning from acids and alkalis for fear of perforating the stomach, there is far greater danger of perforation by leaving the poison in the organ, with the additional risk of cardiac or respiratory failure, or subsequent damage to the intestines or other viscera.

Lavage by siphonage of the stomach is indicated in all cases of poisoning. Warm water at about 101° F. should be employed and the organ washed until thoroughly clean. Any bland fluid, such as tea, coffee, soup, water, or milk, can be used in an emergency to dilute the poison and wash the stomach.

A gastric siphon can be improvised from a fountain syringe by removing the tip and clip and rounding the edges of the extremity; or from a kitchen funnel and rubber tube of small caliber.

If lavage be impossible, then give a pint of lukewarm water with ʒij (8.0) of mustard dissolved therein, or warm salt water and tickle the fauces with the finger or a feather.

Apomorphin, gr. $\frac{1}{10}$ (0.006), by hypodermic; zinc sulphate, gr. 30 (2.0), in ℥iv (30.0) of water; copper sulphate, gr. 5 (0.3), providing these latter were not the poisons ingested; or syrup of ipecac, ʒj to iij (4.0–12.0), can be employed.

Among useful *demulcents* are whites of raw eggs, milk, olive oil, barley-water, flour boiled with water, and gum-arabic water. Fats and oils *should be avoided in phosphorus-poisoning.* The subsequent treatment is of acute gastritis, enteritis, and of special symptoms.

Antidotes.—*For Acid Poisoning.*—Alkalis,[1] such as calcined magnesia; powdered chalk in water; sodium carbonate (washing soda), dilute; potassium cabonate; sodium or magnesium sulphate, ℥ss (16.0), in water; Carlsbad salts; soapsuds in water.

For Carbolic Acid.—Alcohol (95 per cent.), ℥j to iij (32.0–96.0); raw whisky or brandy, or liquor with a large percentage of alcohol. The alkalis can be used subsequently or alone if alcohol or liquors are not obtainable.

For Caustic Alkalis.—Dilute acids, such as dilute vinegar or lemon juice; tartaric or citric acid.

For Tartar Emetic and Antimony and its Compounds.—Tannic acid or strong tea.

For Arsenic and its Compounds.—Tincture of perchlorid of iron, ℥iss (48.0) in a wineglass of water, add ℥ss (16.0) sodium carbonate (washing soda) in half-tumbler of water, mix, and administer. This renders insoluble about gr. 5 (0.3) of arsenic. Repeat dose, or give dialyzed iron, tablespoonful doses.

For Copper Salts.—Potassium ferrocyanid, ʒj (4.0) to ℥iv (125 cc.) of water, forms insoluble copper cyanid.

For Iodin, Iodids, and Iodoform.—Starch solution in cold water; sodium bicarbonate; lead acetate, ʒij (8.0) in ℥iv (125 cc.) of water.

For Lead and its Salts.—Magnesium or sodium sulphate, or dilute sulphuric acid, ♏30 (2.0), in water.

For Mercury and its Salts (Corrosive Sublimate, etc.).—White of raw egg; milk; form albuminate.

For Silver Nitrate.—Salt solution.

For Zinc Salts.—Sodium or potassium carbonate; tannic acid; tea; white of egg; milk.

For Phosphorus (Rat Paste, Matches).—Copper sulphate, gr. 3 to 5 (0.2–0.3), in ℥iv (125 cc.) of water, a number of doses; forms insoluble phosphid of copper and acts as an emetic; old French turpentine. Avoid oils, fats, milk, and yolks of eggs. Avoi American or German turpentine.

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[1] Soda bicarbonate, ℥j (16.0), or plaster scraped from the wall, dissolved in water ℥viij (250 cc.) can be employed.

For Alcohol.—Ammonium carbonate, gr. 3 (0.2), in water.

The stomach should receive rest after immediate treatment Pain may be relieved by local heat, or codein or morphin, gr. $\frac{1}{8}$ to (0.008–0.016), hypodermically. Large doses of bismuth subnitrat may be given if the bowels continue irritable. Retention or suppression of urine must be watched for. In the latter case, enteroclysis, with hot normal salt solution at 115° to 120° F. Stenosi of the esophagus may be dilated if possible, if stenosis of the pylorus appropriate treatment as for gastric dilatation, and in most case subsequent operation. If atrophy of the gastric mucosa result the treatment is for achylia gastrica. For perforative peritonitis operation is indicated.

PHLEGMONOUS GASTRITIS

(*Synonyms.*—Suppurative Inflammation of the Stomach; Gastritis Phlegmonosa Abscess of the Stomach.)

This is a rare disease and usually runs an acute course, thoug occasionally subacute. The process begins in the submucosa an sometimes extends to the muscular coat, and at times to the mucou or serous coats. It is more frequent in men. It is primary or idiopathic, due to some micro-organism, especially the streptococcus probably entering through some solution of continuity in the mucou membrane; or secondary (metastatic), due to pyemia, puerpera infection, or the exanthemata.

Errors in diet, alcoholic excess, trauma, etc., have been given a causes, but probably only are contributory by depressing the system Traumatism might cause damage to the mucosa and render infectio more easy.

There are two forms met with: a diffuse purulent infiltratio and a circumscribed abscess.

Morbid Anatomy.—In the diffuse type a large area of th submucosa is frequently involved. It is thickened, infiltrated wit pus, and multiple small abscesses are often present. The pylori end is more frequently attacked. The muscular wall is often involve or the pus may burrow through to the peritoneum. The mucos is usually also affected and is swollen, and there is granular degeneration of the gland cells. Perforation of the mucosa may occur.

In the abscess type there is generally a single circumscribed abscess of variable size, starting in the submucosa and involving the muscular layer. The mucosa and serosa are often involved. It may terminate favorably by perforating into the stomach (a rare event) or perforate into the peritoneal cavity.

Symptoms.—The patient may have for a brief period a few dyspeptic symptoms, such as loss of appetite, thirst, and some burning in the stomach, but these are usually absent.

The attack is generally of an acute fulminating type: Severe pain or burning in the gastric region, a rapid rise of temperature to 103° to 105° F., with slight intermissions; frequently chills; often vomiting of mucus, bile, and food remnants, but no pus, unless the abscess breaks into the stomach, which is a rare event. The area over the stomach is very sensitive to pressure and there is some tympanites. There is constipation or, more usually, diarrhea. The pulse is rapid and feeble, occasionally there is jaundice.

The patient presents all the aspects of a severe infection, with delirium and coma preceding death. The blood examination shows *leukocytosis* with increase in the *polynuclears*. There is *muscular rigidity* in the *upper quadrant* of the abdomen, of the recti muscles, due to peritoneal irritation even before perforation occurs. This is a valuable sign. With general peritonitis we have the usual symptoms —pain, distention, general tenderness on pressure, etc.

With a circumscribed abscess the tenderness is more localized, the symptoms not so severe, and the duration longer. Percussion and palpation may locate the process if localized.

Duration.—Three or four days, rarely one to two weeks.

Diagnosis.—The temperature, chills, fulminating character, leukocytosis, and early recognition of muscular rigidity—all point to an acute suppurative process. Abscess of the liver and subphrenic abscess are not of such acute type. Acute cholecystitis and acute pancreatitis are more apt to be confounded with abscess of the stomach.

With acute pancreatitis the temperature at first may be low, tympanites is earlier and more marked, and there are circumscribed tenderness in the course of the pancreas and tender spots throughout the abdomen (Fitz). Constipation is usual; also the symptoms may resemble intestinal obstruction. Abscess of the pancreas is slower in its course and there is a longer history.

Acute cholecystitis is a much more frequent condition than phlegmonous gastritis. The gall-bladder can at times be palpated, is tender, and we have the previous history.

Treatment.—Laparotomy is advised, both to settle the diagnosis and afford relief if possible. Should this not be consented to, then the ice-bag, rectal feeding, enteroclysis, to relieve tympanites, and opiates are indicated.

CHRONIC GASTRITIS

(*Synonyms.*—Chronic Gastric Catarrh; Chronic Dyspepsia.)

Definition.—A chronic inflammation of the mucous membrane of the stomach, with the production of mucus and changes in the gastric juice, causing disturbances in the act of digestion.

Etiology.—This disease is more frequent in men than in women. It may follow the acute type, especially after recurrences of this condition. The same irritating agents that cause acute gastritis can produce the chronic type when acting for a long period of time; notably, fast eating with imperfect mastication, overloading the stomach, indigestible food, highly spiced dishes, cold drinks in excess, overrich food, excessive use of tea or coffee, and overindulgence in alcohol (the so-called "drunkards' catarrh"); excessive use of tobacco, especially in tobacco-chewers and those who indulge in dry smoking (chewing the cigar-butt), an unhealthy condition of the mouth or teeth, and swallowing the products of decomposition. Habitual use of drugs may be a cause.

Discharges from the nose or ear through the Eustachian tube, which are then swallowed, have been factors in several cases that I have observed. Thrush may cause mould in the stomach. Chronic gastritis may be secondary to the acute infectious diseases, such as typhoid fever. It is frequently associated with cancer of the stomach and is often present in the atonic type of dilatation of the stomach.

It may be secondary to cirrhosis of the liver, pulmonary or cardiac disease, and chronic nephritis. Among constitutional causes are gout, diabetes, leukemia, and severe anemia.

Classification.—Chronic catarrhal gastritis may be classified as follows:

1. *Acid gastritis* (gastritis hyperpeptica, hypersthenic gastritis), or acid catarrh of the stomach, first described by Boas.

This is considered on the borderline and to be the initial stage of chronic gastritis. There is a slight increase in the hydrochloric acid and Boas believes this occasionally occurs in the early stages of chronic gastritis. There is also the presence of mucus.

2. *Chronic catarrhal gastritis*, under which is described mucous gastritis (Ewald), which is merely a severe type with great secretion of mucus.

We must remember that occasionally from a long-continued chronic gastritis, other conditions may arise.

(*a*) There may be a great thickening of the mucosa (hyperplasia), so as to produce thick folds, the so-called etât mamelonné, and this combined with swelling of the mucosa cause benign stenosis of the pylorus; or sometimes wart-like excrescences (gastritis polyposa) develop, which if situated at the pylorus can produce partial obstruction—a benign stenosis, with dilatation of the stomach; or a proliferation of interstitial tissue occurs and muscular hypertrophy with resulting hypertrophic stenosis of the pylorus.

Chronic gastritis may, therefore, produce and have associated with it benign stenosis and dilatation of the stomach.

(*b*) On the other hand, a degeneration or atrophy of the muscular fibers due to hyperplasia of the connective tissues may cause atony, and atonic dilatation of the stomach may result, and these conditions will improve with the improvement of the gastritis.

They only occur in the more advanced cases. In the patients whom we are called upon to treat, the stomach is *usually of normal* *size*, non-atonic, and with normal or slightly diminished motor functions.

3. *Atrophic gastritis* (Anadenia ventriculi, Ewald), an atrophy of the mucous lining of the stomach, can result from chronic gastritis. Of these, there are two forms:

(*a*) Phthisis ventriculi, a thinning of the coats of the organ, which may remain of normal size or be increased in size.

(*b*) Cirrhosis, or sclerosis ventriculi, an enormous thickening of the muscular coat and a great reduction in the volume of the stomach.

Atrophic gastritis is described under Achylia gastrica.

Morbid Anatomy of Chronic Catarrhal Gastritis.—The mucous membrane is yellowish-gray or slate-gray in color, and in secondary catarrhal conditions produced by congestion may, in some parts, be intensely red. It is swollen and thickened and covered with a closely adherent tenacious mucus, which is usually cloudy and gray in color, due to various cells, epithelia, and leukocytes, and occasionally is tinged with blood. Enlarged veins, patches of ecchymosis, and small hemorrhagic erosions may be present. The mucosa in some instances forms papillary projections (etât mamelonné).

The pyloric portion is usually involved, though the inflammation may extend over the entire mucosa. The submucosa and muscular coats are at times hypertrophied or atrophied.

Microscopically the following conditions appear: The glands are enlarged, sacculated, and dilated in cyst-like forms, the tubuli in many places being atypic and branching like the fingers of a glove; the gland-cells are cloudy, granular, and in a condition of fatty degeneration, and the principal and parietal cells cannot be differentiated. Abundant small cell infiltration presses the glands apart, being especially marked toward the surface of the mucosa. Extensions of connective tissue may be seen passing from the mucosa between the glands. The mouths of the glands are at times filled with mucus which projects against the lumen. Mucoid transformation of the cells of the tubuli is a striking feature and may extend to the fundus of the glands and cells. Mucoid degeneration may replace the principal and parietal cells.

The mucus fills part of these cells, or may crowd the protoplasm and nucleus against the base, or rupture the cell-membrane and escape into the duct of the gland. The pyloric region is usually thus affected. Ewald has described this condition.

After a long period the inflammatory process may lead to a total destruction of the glandular layer, or atrophy of the mucous membrane of the stomach—anadenia ventriculi (Ewald). Of this there are two forms, as heretofore noted:

(*a*) Phthisis ventriculi, atrophy of the stomach, or anadenia ventriculi (Ewald), is a thinning of the coat of the stomach, with a retention of or usually an increase in the size of the organ (dilatation). There are fatty degeneration and destruction of the glands, the process progressing from the surface of the stomach inwardly. In the early stage no glands are found, but glandular cysts are present near the submucosa. Later these disappear and the mucous membrane consists chiefly of round cells. The submucosa is changed and the muscular layer is thinner.

(*b*) In cirrhosis, or sclerosis ventriculi, the stomach coats are thickened and there is a great reduction in the volume of the organ; in some cases it is no larger than a pear and the walls may be 2 to 3 cm. in thickness, the greatest increase being in the submucosa, *where the process starts.* The inflammatory process causes the formation of fibrous tissue, which progresses from the submucosa to the surface, spreading around the glands, constricting them, and finally replaces them with fibrous tissue. The hypertrophy extends to the muscular layer.

This condition may coexist in the cecum and colon and so be difficult to distinguish from diffuse carcinoma. Proliferative peritonitis with perihepatitis and ascites are at times associated.

Atrophy of the gastric mucosa is described under Achylia gastrica.

Symptoms of Chronic Gastritis.—These develop slowly; the appetite diminishes or is irregular or easily satiated. There is a feeling of fulness or pressure in the gastric region after eating. Occasionally *heartburn* or cardialgia in the epigastrium or precordial region or behind the sternum occurs, generally *at the height of digestion;* while with hyperchlorhydria it is present *on an empty stomach.* Discomfort or pain on pressure over the stomach is present. *Belching of gas is the most frequent symptom* and it is usually odorless; waterbrash (pyrosis), a bitter or a tasteless fluid, may be brought up into the mouth (regurgitation); the stomach and intestines are often markedly distended with gas. Nausea is frequently present and occasionally vomiting.

When the latter occurs it is frequent in the morning, when the stomach is empty, and consists chiefly of slimy mucus, and sometimes of partly digested food of the previous day with mucus. It may take place after breakfast. There is a sour, bad, or salty taste in the mouth. The patient in some cases complains of palpitation and shortness of breath (dyspeptic asthma or, more correctly, dyspnea). The pulse is small and sometimes slow. There are fulness in the head, insomnia, lack of energy, and distaste for work. Dizziness may be present. Sensation of fear, depression, melancholia, or hypochondriasis occur in some patients. The tongue is usually covered with a thick gray moist fur, though not always so, and it cannot be said to be characteristic. The margins are at times indented. Odor of the breath is present when there is disease of the mouth or teeth, or atony of the stomach with fermentation. Headache is quite common and a desire to yawn. There is at times the so-called stomach cough, doubtless due to pharyngeal irritation.

The patient's appearance may be quite good and he may preserve his weight. In severe cases he looks quite badly, and shows black rings under his eyes, has cold hands and feet, and chills easily. Some even lose considerable weight and become thin and emaciated. In the severe types with nervous symptoms, intestinal fermentation or putrefaction are often present and *auto-intoxication* is undoubtedly a factor. The bowels are, as a rule, *constipated;* though occasionally diarrhea or diarrhea alternating with constipation are present.

Physical Examination.—*Inspection.*—The gastric region appears bloated.

Percussion.—Tympanites is present, but the stomach is usually in the normal position.

Palpation.—The gastric region is sensitive to pressure; tenderness is rather diffuse. No real pain or sense of resistance.

Splashing sound can be produced if liquid and gas are present. It is only abnormal if found at a time *when the stomach should be empty.* It would then show atony. If found in an *abnormal position* (low), it is an evidence of dilatation or ptosis. With movable kidney ptosis can be diagnosed.

Urine is scanty; contains phosphatic and urate deposits. Specific gravity is increased.

Diagnosis.—The *presence of gastric mucus* in the stomach contents is the *chief diagnostic point* in chronic *gastritis*, so that examination of the *vomitus* or, *preferably, of the gastric contents* after a *test breakfast is imperative.* The *diagnosis should not be made* from *clinical symptoms alone.*

Gastric Contents.—One hour after Ewald's test breakfast or the one I employ—2 slices (60 gm.) of bread without butter and 250 to 300 cc. of water—the latter being slightly less than Ewald's, the contents of the stomach are withdrawn. The following are the conditions found present:

Total acidity is diminished; free hydrochloric acid is markedly lessened or is small in amount or absent; pepsin and rennet are present but diminished; erythrodextrin present in small quantities; achröodextrin and sugar abundant; quantity of gastric contents frequently normal (under 100 cc.) or may be slightly more (100 to 150 cc.), which last would show some motor insufficiency.

The pieces of roll are not as fine as normally, but larger and coarser. Mucus is usually intimately mixed with the food remnants and is adherent to the morsels of food. The stomach contents are thick, tough, and sticky, and difficult to filter. A glass rod dipped into them and lifted up will draw up strings of mucus with it. Acetic acid added to the filtrate produces turbidity.

Mucus that has been swallowed is never mixed with the food remnants, but floats as isolated balls on the surface.

Mucus in some cases is in large amount, while there may be very little in others. In the latter event, lavage of the empty stomach will determine its presence, in shreds or flakes.

In the fasting condition there are often only a few cubic centimeters of turbid liquid in the stomach, consisting chiefly of mucus of an alkaline, neutral, or slightly acid reaction. If no contents can be thus secured, lavage again will show the mucus.

The vomitus shows the same characteristics already described, but the examination by the test breakfast is more accurate.

Microscopically.—Mucus, round cells, and epithelial cells are found to be present. In doubtful cases the microscope may differentiate the types of mucus.[1] If squamous epithelia be mixed with it, it probably comes from the mouth or pharynx; if pigmented alveolar epithelia, probably from the air-passages.

In *acid gastritis*, we find the total acidity and free hydrochloric acid slightly increased, and the presence of mucus. I had recently a case in which the total acidity averaged 90+ and free hydrochloric acid 70+, with a large amount of mucus.

The so-called cases of mucous gastritis merely contain an excessive amount of mucus, with little or no hydrochloric acid.

In atrophic gastritis there is absence of hydrochloric acid, absence of pepsin and absence of rennet, as described under Achylia gastrica.

If dilatation be present, we have the physical signs and gastric findings of such.

Einhorn finds small shreds of the mucosa present in the wash-water of some cases of chronic gastritis due, as he believes, to erosions.

Motor function may be *normal* or slightly diminished, so that the ingesta escape before fermentation can occur. This is the usual course in the ordinary type of case.

In some cases with hypertrophy the motor function may be increased. If atony or dilatation is present, there are motor insufficiency and fermentation.

In those with excessive mucus production, the action of the saliva and gastric juice is interfered with and though the motor function is good, the ingesta passes into the intestine with little change, and intestinal fermentation or putrefaction results.

Absorption.—This depends on the severity of the case; in milder cases, with the iodid of potassium test, it seems normal; in severe cases it is interfered with.

Course.—The duration of chronic gastritis is long, often extending over many years. Marked improvement may take place, but relapses are apt to occur from indiscretions. Milder cases can be permanently cured.

Differential Diagnosis.—*Chronic Gastritis.*—No severe pain; no circumscribed spot painful to pressure; no hematemesis; no cachexia; no marked emaciation, except in severe cases of long duration; free HCl diminished or absent; gastric mucus present; slow course.

Ulcer of the Stomach.—Hyperchlorhydria, as a rule; severe pain in the epigastrium with intervals free from pain when the stomach is empty; local tenderness which is circumscribed; dorsal pain, hematemesis, or occult blood in the stool or gastric contents; no mucus; patient has appearance of suffering; no true cachexia.

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[1] Columnar epithelia mixed with mucus show it is gastric.

Cancer.—Age (usually over forty-five); *rapid course;* free HCl usually markedly diminished or absent; lactic acid present; mucus sometimes present; pain generally continuous, but not as acute as in ulcer; Boas-Oppler bacillus; cachexia; tumor on physical examination; small amount of blood present in gastric contents; blood or occult blood in the stool; hematemesis much less than ulcer; foul odor to vomitus at times present.

Achylia Gastrica.—Slow course; scarcely any gastric juice; acidity very low (2 + to 4 +); absence of HCl; absence of pepsin; absence of rennet; usually *no mucus;* no lactic acid. In the early stage (transitional) Riegel holds that mucus may at times be present.

Gastric Neuroses.—Symptoms not uniform; character of food makes little difference; indigestible food may be well borne and digestible food may cause symptoms; *no gastric mucus;* HCl may be diminished or in some cases normal or increased, and the gastric findings often vary at different times in the same patient. *Subjective symptoms are sometimes similar to chronic gastritis,* but they disappear and reappear abruptly.

Prognosis.—Some cases can be cured; many improved. Relapses may occur. The affection is frequently a tedious one.

Treatment.—This may be divided into: (1) Prophylaxis. (2) Hygiene. (3) Local treatment of the stomach. (4) Diet. (5) Mineral springs. (6) Medication.

Acid Gastritis.—The borderline cases, *acid gastritis,* should receive the treatment of hyperchlorhydria; also occasional lavage to remove mucus, say, twice a week, is advisable. In a recent case I have employed it daily, using several ounces of milk of magnesia (Phillips) to the quart of water.

Extract of belladonna, gr. ⅓ (0.022), t. i. d. before meals and magnesia usta and soda bicarb., āā, gr. 10 (0.6), combined with resorcin resub., gr. 5 (0.3), in water, an hour after meals, as suggested by Theodorus Bailey, is excellent treatment. Milk of magnesia, ʒj to ij (4.0–8.0), is also of value.

Van Valzah and Hayes report cases.

1. *Prophylaxis.*—Unquestionably, repeated attacks of mild so-called acute dyspepsia (acute gastritis) may ultimately lead to the development of chronic gastritis. The causes of both conditions are practically the same. The patient, therefore, should masticate thoroughly, avoid bolting the food, overindulgence in alcohol, tobacco, very hot or very cold food and drink, indigestible food, etc. He should rest for fifteen to thirty minutes after meals before returning to business. The mouth and teeth should be kept in good condition; and if there are aural or nasal discharges escaping into the mouth, or tonsillar or pharyngeal inflammation, treatment should be instituted.

Cardiac disease should be treated with cardiac stimulants, especially if there is failing compensation; and diseases of the liver an kidney should receive appropriate diet and treatment, so as t lessen the chances of secondary gastritis.

2. *Hygiene.*—Slow eating, with subsequent rest; exercise, preferably in the open air, driving, golf, rowing, walking, and horseback—all of which should be carried out in a leisurely manner and no overdone, so as to exhaust the patient, are useful. Moderate gymnastic exercises five or ten minutes daily, as with an exerciser, wit open windows, are of value.

Avoid badly ventilated rooms and sleep with the windows open

Cold salt-water sponging morning and night, followed by frictio with a rough towel, is of service.

If the patient is excessively nervous, change of climate may b necessary.

3. *Local Treatment.*—The removal of the mucus is of importance This can be done by two methods: By administering alkalin remedies that will dissolve the mucus and by lavage.

In mild cases lavage is not always necessary and the use o alkalis, as advocated by Achilles Rose, is of service.

They should be administered about an hour before breakfast and if necessary, also before luncheon and dinner. For example, in ℥v to viij (200 to 250 cc.) of hot water, soda bicarb., gr. 30 to ʒj (2.0-4.0), or lime-water, 16.0 (℥ss), or milk of magnesia (Phillips), 4.0 t 8.0 (ʒj–ij).

Magnesia usta, 2.0 to 4.0 (gr. 30–ʒj), is also of use, alone o combined with equal quantities of soda bicarbonate.

Penzoldt has demonstrated that mucus will adhere to bismuth Rose takes advantage of this and gives a tablet consisting of 0.6 (gr. 10) magnesia usta and 0.6 (gr. 10) bismuth subnitrate, to b chewed with a full glass of water one hour before meals; or two tablets, if required.

The magnesia usta, milk of magnesia, or soda bicarb. are especially useful.

Lavage.—When the mucus secretion is more marked, lavage should be substituted; or if a mild case does not improve by the above method. This should be employed on the empty stomach before breakfast, so as to aid subsequent digestion. An alkali should preferably be added to the fluid to dissolve the mucus. The stomach should be washed with the patient both sitting and lying down and turning on the sides, to remove all mucus possible, and washed *until the outflow is clear.*

The following are excellent: Milk of magnesia (Phillips), 30.0 to 60.0 (℥j–ij) to the liter (quart); or lime-water the same quantity; or soda bicarb., 4.0 to 8.0 (ʒj–ij); or magnesia usta, 4.0 to 8.0 (ʒj–ij)—all to the liter (quart).

Normal saline solution—ʒj (4.0) salt to water Oj (500 cc.)—or boric acid, 4.0 (ʒj) to the liter (quart), may be employed.

I use normal saline solution combined preferably with milk of magnesia or magnesia usta.

Fleiner mixes 2 parts sodium chlorid and 1 part soda bicarb. and employs 4.0 (ʒj) to 2 to 3 liters of water.

Frequency of Lavage.—Once a day before breakfast is often sufficient; in some cases it may be necessary to repeat before supper.

If dilatation with fermentation, resorcin (resub.), 0.3 to 1.0 (gr. 5–15), or the same quantity of salicylic acid or sodium salicylate; or gomenol, 1.0 to 2.0 (15 drops–ʒss); or potass. permang., 0.1 to 0.3 (gr. 1½–5); or listerin, glycothymolin, or borolyptol, 4.0 (ʒj)—all to the liter (quart), can be employed.

In such event, I wash with the alkali in the morning to remove mucus, and with the antifermentative at night.

In some cases, lavage with nitrate of silver 1:5000 to 1:2000 is of value, used every two or three days, preceded by warm water lavage, to first remove mucus. No saline should be used in the silver solution. Saline solution may subsequently be used if the silver cause pain or irritation.

Pepper, in place of this, advocates an aqueous solution of silver nitrate, dose, gr. ⅛ to ¼ (0.008–0.016), internally three times a day for a short period. This should be kept in a dark bottle.

Electricity.—Unless atony with lessened motor function of the stomach, or severe subjective symptoms, with *little mucus* are present, electricity is of no practical value.

When there is little mucus, it may aid to stimulate gastric secretion. In such event the intragastric method is preferable to the percutaneous. Removal of the mucus is of the first importance. Electricity, however, is of service applied to the abdomen to *increase intestinal peristalsis.*

Massage.—Vibratory Massage.—The same holds true of these methods. They are also of value over the intestines to promote peristalsis.

Hydrotherapy.—In sensitive cases hot water compresses applied to the stomach are useful.

4. *Diet.*—This is an *important feature*, and its character depends on the severity of the symptoms. It is always preferable to give four or five light meals than three full meals a day. In severe cases give food in liquid and semiliquid form (mushes), such as milk or matzoon with Vichy, kumyss, bacillac, lactone-buttermilk, barley, oatmeal, or rice soups prepared with milk; or chicken soup or bouillon, with raw egg beaten up; sanatogen (plain or flavored), somatose, plasmon, or tropon can be added to the soups.

Later, soft-boiled eggs, mashed potatoes, rice gruel, scraped raw or broiled beef, toast baked in the oven, stale bread (white), butter, cocoa, and weak tea.

The diet should be mixed, the albumin somewhat reduced; th carbohydrates should be given in suitable form, avoiding those tha contain too much cellulose. Fats, such as butter and cream, ar especially necessary when there is *loss of nutrition.* In the latte case I feed by the "scales," endeavoring to put on weight, improv assimilation, and at the same time ameliorate symptoms.

The mere estimate of requisite calories and feeding by thi method *alone* is of no value, as each patient is a rule to himself.

For example: In severe cases, at first liquids and mushes.

8.00 A. M. Milk with ℥ij (about 125 cc.) of lime-water, or peptonized milk, ℥viij (about 250 cc.), with sanatogen, ʒj (4.0).

10.30 A. M. Matzoon and Vichy equal parts, in all ℥vj to vii (200–250 cc.).

1.30 P. M. Oatmeal soup or chicken soup, with an egg beaten in ℥viij (250 cc.).

4.00 P. M. Same as at 10.30 A. M.

6.30 P. M. Same as at 8.00 A. M.

9.30 P. M. Milk and Vichy equal parts, in all ℥viij (250 cc.).

Additions can gradually be made to this diet. Milk, however does not agree with some, and soups and broths must be substituted

The following diet is useful in many cases for a week or two but must be modified to suit the individual.

		Calories
8.00 A.M.	1 cup cocoa or tea, about two-thirds milk, approximately	10
	1 lump of sugar	4
	2 soft-boiled eggs	16
	2 ounces zwieback, or toast, or stale white bread (2 slices)	15
	½ ounce butter	11
10.30 A.M.	8 ounces koumyss, matzoon, or milk (250 cc.)	16
	2 ounces crackers or somatose biscuit	15
	½ ounce butter	11
1.00 P.M.	2 ounces of steak, chicken, or chop	7
	3 ounces of mashed potatoes or rice	13
	2 ounces white bread (stale), or toast, or zwieback	15
	1 cup tea, about two-thirds milk, approximately	10
	½ ounce butter	115
4.00 A.M.	7 ounces milk, mixed with 1 ounce top-cream (250 cc.)	210
	1 ounce crackers	100
	½ ounce butter	100
6.30 P.M.	8 ounces hominy, rice, or cereal boiled in milk (250 cc.)	450
	2 scrambled or poached eggs	165
	2 slices bread average about 2 ounces	150
	½ ounce butter	115
		2858

The above is for about three weeks, and the diet of each patient should later correspond, as nearly as possible, to the usual mode of living.

There are things which it is necessary to forbid, such as fried food; meat with tough fibers or that is too old or too fresh, pork, sausages; lobster, salmon; chicken salad; mayonnaise; cucumbers, pickles, corn, radishes, celery, cabbage; hot breads, brown and

Graham bread; also fresh bread and *all* alcoholic beverages, which last I believe do special harm to the inflamed mucous membrane.

Foods which disagree should be interdicted. Sugar should be taken in small quantity and avoided by some. Soda-water and candy are forbidden; also hot and cold drinks and ice-cream. Veal, as a rule, in this country is interdicted, as it is often tough.

Salt is of value, as it aids the production of HCl.

Beef, mutton, lamb, chicken, potatoes, hominy, rice, oatmeal, spinach, lettuce, asparagus, eggs, etc., are all admissible. Water should be taken in small amounts during the meal.

Smoking.—Excessive smoking should be stopped. It is chiefly the *tobacco juice* from chewing the cigar, carried by the saliva, that damages the mucous membrane of the stomach; chewing tobacco should be forbidden.

If a cigar-holder or cigarrette-holder be employed, I can see no objection to two cigars or four cigarettes a day; otherwise it should be interdicted. If there is nasopharyngeal catarrh, smoking should be stopped.

5. *Mineral Waters.*—These dissolve the gastric mucus, hasten the emptying of the stomach, and often stimulate the mucosa. On account of the rest and regular life and diet the springs are often preferable, though the waters may be taken at home. The most useful are the saline and the saline-alkaline waters.

(*a*) *Saline Springs.*—These contain chiefly sodium chlorid and varying quantities of carbonic acid gas, and stimulate the secretion of hydrochloric acid. The most notable are Kissengen, Homburg, Wiesbaden, Soden, and Saratoga (Congress Spring). Dose, glass of mineral water on arising.

(*b*) *Alkaline-saline Springs.*—These contain sulphate of soda, sodium bicarbonate, sodium chlorid, and carbonic acid gas.

The Carlsbad Springs are the most famous. Marienbad and Saratoga (Hawthorne Spring) belong to this group, also Glauber's Salt Springs and Glauber's salts.

One can employ the imported Carlsbad salts or Glauber's salts; and by adding sodium bicarbonate to Glauber's salts imitate imported waters more closely.

Wolff's formula for artificial Carlsbad salts:

℞.	Sulphate of soda	30.0
	Sulphate of potassium	5.0
	Sodium chlorid	30.0
	Carbonate of soda	25.0
	Biborate of soda	10.0.—M.

Sig.—℥ss to j (2.0–4.0) in warm water before breakfast.

These springs are of value for the solution of large quantities of mucus and for constipation. Care should be taken not to purge the patient excessively, and nervous cases do not take them especially well.

6. *Medication.*—The methods described will often be sufficient, but medication is of service as an accessory.

Leube was the first to recommend the use of dilute hydrochloric acid[1] to supplement this deficiency of the gastric juice. Ewald advised the use of large amounts, 40 to 60 drops, taken in divided doses three times a day after meals.

For example: The entire dose in a glass of water and commencing one-half hour after meals, a third of this being taken every fifteen minutes.

I prefer a smaller dose, from 15 to 20 drops, in a small glass of water three times a day one-half hour after meals, and taken in three divided doses, as advised by Ewald. This is impossible to pursue with some patients, as they will not devote the time; so in such an event a single dose can be administered, commencing at 5 drops and gradually increasing to 15 drops.

The following is an excellent prescription:

℞.	Tinct. nucis vomicæ		12.0 (ʒiij)
	Acid. hydrochlor. dilut. Comp. tinct. cinchona	āā	16.0 (℥ss)
	Aq. destil.	q. s. ad.	125.0 (℥iv).—M.

Sig.—4.0–8.0 (ʒj to ij) t. i. d. in water one-half hour after food.

Pepsin is present in considerable quantity in chronic gastritis, so it is hardly indicated; though some add 0.5 to 1 (gr. 7½–15) in combination. Papayotin, papain or papoid, or pancreatin, 1.0 to 1.5 (gr. 15–22½), with sodium bicarbonate, have been suggested; also the diastase combinations, wines of pepsin, etc.

The use of artificial digestants would tend to weaken the gastric functions, and are not indicated.

The bitter medicaments as stomachics, to stimulate the function and appetite, are often employed, and Riegel believes the hydrochloric acid acts in this way. They should be used fifteen minutes before meals in 1 to 2 ounces of water.

Among such remedies are tincture aurantii amara; tinct. amara (bitter tincture, Squibb's); tinct. calumba; fluidextract calumba; tinct. cardamomi; tinct. hydrastis; tinct. gentian comp.; fluidext. hydrastis; fluidext. condurango; fluidext. quassia.

The average dose for this purpose of any of these remedies would be from 15 to 20 drops; quassia, in 5 to 10 drops; and tincture nux vomica, if given as a stomachic, in 5 to 10 drops.

They may be given alone or in combination, with smaller individual doses.

The alkaloidal form of administering stomachics is often of value. Thus:

Condurangin (Abbott's), gr. $\frac{1}{67}$ (.001).

Quassin (Abbott's), gr. $\frac{1}{67}$ to $\frac{1}{12}$ (.001–.005).

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[1] Oxyntin, a derivative of HCl, combined with nux (tincture), 5 drops, given in capsules, gr. v (0.3), after meals, is of value.

Condurangin (Merck's), gr. $\frac{1}{10}$ to $\frac{1}{4}$ (.0065–.016).

Quassin (Merck's), gr. $\frac{1}{32}$ to $\frac{1}{3}$ (.002–.02).

Hydrastin (Merck's), gr. $\frac{1}{4}$ to $\frac{1}{2}$ (.016–.032).

Strychnin arsen., gr. $\frac{1}{100}$ (0.00065), and quassin, gr. $\frac{1}{10}$ (0.0065), are a good combination.

Orexin, gr. $\frac{1}{2}$ to 1 (0.032–0.065), can be given in bouillon for the same purpose.

In the gastritis of phthisis, carbonate of creosote or guaiacol carbonate, gr. 5 (0.3) each t. i. d. after meals, are of value.

For Nausea and Vomiting.—Cerium oxalate, gr. 1 (0.065), alone or combined with soda bicarb. or bismuth subnitrate, gr. 2 (0.13), or any of the methods described under Acute Gastritis. Lavage may be necessary.

For Belching.—Milk of magnesia, ʒss to j (2.0–4.0), or magnesia usta, gr. x (0.6).

For Gas and Intestinal Fermentation.—Resorcin resub., ʒiss (6.0); Aq. menth. piperit., q. s. ℥iv (125.0). Dose, ʒij (8.0) t. i. d. after food in water, or ichthoform or ichthalbin, gr. 5 (0.32) each, etc.

For *nervous symptoms associated with intestinal putrefaction* I would refer to the chapter on the latter subject. Iron and arsenic can also be added to the treatment.

For Constipation.—Patients under treatment with the Carlsbad waters require no treatment for constipation; otherwise attention must be paid to this condition.

The patient should have a movement every day, go to the closet at a definite hour, and endeavor to secure bowel action. This can be assisted by a 2-ounce injection of olive oil, or by a glycerin or gluten suppository, or ʒij to ℥ss (8.0–16.0) glycerin in ℥j (30.0) of water, by means of a small rectal syringe.

Green vegetables, such as spinach, asparagus, lettuce, green peas, etc., are of service.

Stewed fruits, such as prunes, apples, or pears, are often effectual.

Some cases do well with food containing much cellulose and with rye bread, but many cannot take them. A glass of cold or hot water on rising is of value.

If mild cathartics are necessary, cascara (fluidextract), ʒss to j (2.0–4.0), or the aromatic fluidextract; or extract cascara, gr. 1 to 2 (0.065–0.13), or compound rhubarb pills; all at night.

The following prescriptions are of service.

℞. Aloin........................gr. $\frac{1}{4}$ (0.016)
Podophyllin....................gr. $\frac{1}{6}$ (0.011)
Atropin. sulph. }
Strychnin } ....āā gr. $\frac{1}{100}$ (0.00065)
Cascara ext. ....gr. $\frac{1}{4}$ (0.016).—M.
In one pill.

℞. Ext. aloes }
Ext. nucis vomicæ } ....āā gr. $\frac{1}{8}$ (0.008).—M.
Ext. belladonnæ }
In one pill.

℞. Podophyllin....................gr. $\frac{1}{6}$ (0.011)
Ext. nucis vomicæ } .... āā gr. $\frac{1}{4}$ (0.016).—
Ext. physostig. }
In one pill.

℞. Aloin........................gr. $\frac{1}{4}$ (0.016)
Strychnin.....................gr. $\frac{1}{60}$ (0.00108
Ext. belladonnæ...............gr. $\frac{1}{8}$ (0.008).-
In one pill.

Other remedies are described under Constipation.

Olive-oil injections to be retained at night; an occa of soapsuds, not over 1 quart (liter); massage or vibra or electricity to the intestines are useful.

CHAPTER XI

ACHYLIA GASTRICA

(*Synonyms*.—Atrophy of the Stomach; Atrophy of the Gastric Mucosa; Anadenia Ventriculi; Phthisis Ventriculi; Atrophic Gastritis; Catarrhus Atrophicans.)

Definition.—Achylia gastrica (the term first suggested by Einhorn) may be defined as a functional perversion of the stomach, characterized by the absence of the gastric secretion (of hydrochloric acid, pepsin, and rennet).

Introduction.—In many cases, atrophy of the mucosa is the cause, and the condition is permanent. Einhorn, however, has reported a case in which there was eventually some return of secretion, so that portions of the mucosa could not have been much altered; and a case of achylia in a vegetarian, apparently an atrophy from disuse. Achylia gastrica may result from organic changes in the stomach, or may be a pure neurosis, so I prefer to place it in a special chapter.

The *loss of function may be temporary from nervous disturbances*, and A. Rose[1] and the author[2] have observed achylia occurring in gastroptosis, with a return of secretion following the cure of ptosis. Temporary achylia is present occasionally in mucous colic.

History.—Atrophy of the gastric mucosa was first described in connection with pernicious anemia by S. Fenwick,[3] and later by Lewy,[4] Ewald,[5] Osler,[6] Kinnicutt,[7] Nothnagel, Boas, and others. It was believed to be the cause of pernicious anemia and productive of the fatal result. Herter has demonstrated the influence of intestinal putrefaction, chiefly through the gas bacillus (Bacillus aerogenes capsulatus), in the production of pernicious anemia, and favorable results produced by intestinal irrigation and lactic acid fermented milk diet. Stockton has called to our attention that achylia does not occur in the early stages of pernicious anemia, but only when it becomes severe. In view of these facts, achylia seems to be a secondary and not a primary cause.

Numerous non-fatal cases of achylia have been reported by

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[1] Atonia Gastrica, Rose and Kemp.

[2] Observations on Dilatation of the Stomach and Gastroptosis, Medical News, August 6, 1904. Mucous Colic, American Medicine, March 4, 1905.

[3] Lancet, July, 1877.

[4] Berlin. klin. Wochenschr., 1887, No. 4.

[5] Ibid., 1886, No. 32.

[6] American Journal of the Medical Sciences, vol. xci, 1886, p. 498.

[7] Ibid., 1887, p. 419.

Ewald,[1] Boas, Einhorn,[2] Jaworski, Jones, and Martius, and the latter has written a work on the subject.

Etiology.—Severe chronic catarrhal gastritis, or toxic gastritis, may produce permanent destruction of the glands; atrophy from disuse, as in vegetarians; achylia, associated with cirrhosis of the liver (syphilis); or with carcinoma of the stomach, or, rarely, with carcinoma of other organs; occasionally with diabetes mellitus; achylia with pernicious anemia. Organic changes are present in these cases. Achylia may occur *as a temporary functional disturbance* in nervous conditions, neurasthenia, gastroptosis, and mucous colic. Achylia may also be secondary to a general infection, such as typhoid fever,[3] etc.

It is quite common in the middle and later years of life, but has occurred in a number of young persons, especially in the transitory type.

Morbid Anatomy.—When the achylia is of nervous origin, or associated with gastroptosis or mucous colic, there are no organic changes in the mucosa, and the condition is a temporary functional disturbance of secretion. Rose and myself believe the achylia in mucous colic due to the gastroptosis and not to the nervous condition.

Einhorn has shown that in cases where a portion of the mucous membrane has been aspirated, showing the organic changes of achylia, there has been some return of secretory function under treatment, so that this is no evidence of permanent destruction of secretory power in all the gastric glands.

Of the organic changes, there are the two types described under the terminal stage of chronic gastritis:

1. Phthisis ventriculi—round-celled infiltration and fatty degeneration, with thinning of the stomach wall; round cells taking the place of the glands; and the stomach is normal in size or may be dilated (Fig. 126).

2. Cirrhosis, or sclerosis ventriculi, a fibrous inflammation, starting in the submucosa; fibrous tissue takes the place of the glands, and the stomach is contracted and the walls thickened.

Symptoms.—One can scarcely say that there are symptoms characteristic of achylia gastrica; it is *the examination of the gastric contents* which will alone determine the true diagnosis.

In describing the symptoms it seems best to classify achylia under certain groups, some of which we may dismiss briefly.

1. Gastroptosis (splanchnoptosis), with its symptoms; gastric disturbances; achylia present in some cases.

2. Mucous colic, with its symptoms; occasional presence of achylia.

3. Patients with no symptoms and enjoying good health.

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[1] Berliner klin. Wochenschr., 1892, Nos. 20 and 27.

[2] New York Med. Presse, Sept., 1888.

[3] Stockton, American Journal of the Medical Sciences, Aug., 1909.

Einhorn calls attention to this class, one of which had the habit of rumination, and achylia was found present, of forty years' duration. Patient had no other symptom. Clinically this class cannot be considered.

4. Cases with gastric symptoms of varying severity, associated with intestinal disturbances. *These are the most common type.*

There are loss of appetite, a feeling of fulness or pressure in the epigastric or gastric regions, and in some cases severe paroxysms of pain, usually soon after eating and persisting for some time; vomiting may occur soon after the ingestion of food; *belching of gas;* headache and occasionally vertigo; usually constipation.

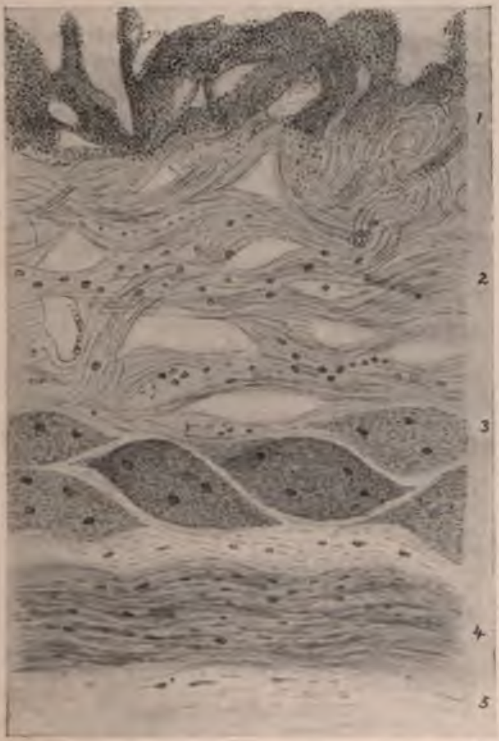

Fig. 126.—Achylia gastrica: 1, mucosa; 2, submucosa; 3 and 4, muscularis; 5, serosa; section shows round-celled infiltration of mucosa and absence of glands.

Some cases may remain fairly well nourished. In others there may be considerable loss of weight, which extends over a period of several years, and nervous symptoms may be present.

In cases in which nutrition is preserved, the intestines perform the digestive functions of the gastro-intestinal tract.

Einhorn describes cases whose symptoms resemble hyperchlorhydria, with pains one to two hours after eating, which are relieved by food and drink, especially the latter, which prevents irritation of the mucosa by the coarse particles. This has been given as an explanation for the pain.

5. Cases with marked intestinal disturbances, especially diarrhea, or occasionally diarrhea alternating with constipation; quite frequently there may be no gastric symptoms or slight belching or feeling of pressure.

Some of the cases lose considerable weight and strength and feel weak. I have recently treated a case suffering from these symptoms who was, in addition, markedly nervous. At the end of four months' treatment gastric secretion returned and all symptoms disappeared, there being 15 pounds increase in weight—a case of nervous achylia.

Some cases of this type may present symptoms (subjective) of diabetes, according to Einhorn.

6. Cases with severe anemia (described by Riegel) with diarrhea. Examination of the blood shows secondary anemia, and of the stomach, achylia gastrica. Diarrhea favors the production of anemia and the latter improves under treatment. These cases must not be confounded with pernicious anemia. The blood examination easily differentiates.

7. Achylia developing during acute febrile conditions, such as influenza (Ewald). Riegel believes it probably pre-existed, and that the intestines, which formerly performed the digestive functions, are thus disturbed and achylia symptoms first appear in consequence.

8. Patients suffering from nervous conditions, or neurasthenia, or gastric neuroses; achylia a temporary perversion.

My case under the diarrheal class belonged to this type.

9. Pernicious anemia with achylia, in which the blood findings are typic; megalocytes and nucleated red blood-corpuscles (normoblasts and megaloblasts), etc.

Diagnosis.—The diagnostic feature of achylia is the gastric analysis one hour after Ewald's test breakfast. The characteristics are as follows:

Total acidity is 2+ to 4+, or even neutral; HCl = O; Pepsin = O; rennet = O (rennet zymogen may be present); propeptone = O; peptone = O; lactic acid = O; or faint trace; erythroödextrin = O; sugar = +; gastric contents have no odor, no evidence of fermentation; quantity of liquid is very small, aside from that soaked into the particles of bread (this is quite characteristic); bread particles are not minutely minced, but rather coarse; absence of mucus.

Schmidt and Riegel believe mucus to be present in some cases in the early stage of achylia, developed from chronic gastritis, while there are some glands remaining in mucoid degeneration, and that the presence of mucus does not signify that the case is not one of achylia. Some cases of old chronic gastritis certainly seem on the borderline.

The small amount of fluid in achylia is explained by the fact that the liquid chyme rapidly leaves the stomach, leaving the solid particles, and that practically no secretion takes place in the stomach.

Motor function is often normal or even increased. It is diminished where there is degeneration and dilatation of the stomach.

Absorption.—Though this has apparently seemed normal by the iodid of potash test, in some cases it is evident that this test is often fallacious, in view of the fact of the general loss of nutrition in some patients.

I agree with Riegel that from the pathologic condition present, absorption must be diminished.

Course and Prognosis.—In nervous achylia, secretion may be resumed in a few months under proper treatment, and in gastroptosis and mucous colic the condition is dependent on the treatment of these diseases.

In some patients the condition perhaps exists for years, with the patient in good health; while in others the subjective symptoms may be removed or cured, while the objective symptom (the analysis) persists. Others run a long and protracted course.

With pernicious anemia and carcinoma the prognosis of achylia depends on the primary disease.

Diagnosis.—Repeated analysis of the gastric contents are necessary to arrive at the diagnosis.

	Achylia Gastrica.	*Cancer of the Stomach.*
Gastric contents:	Little fluid, no mucus, low acid reaction (2+ to 4+), no HCl, no lactic acid, coarse particles, no blood.	Mucus, acidity higher, lactic acid, free HCl may be present, though usually absent, contents more fluid, and odor and food less coarse, blood.
Stool:	No blood.	Blood, or occult blood.
Tongue:	Often clean.	Coated.
Pain:	At times.	Constant.
Motor power:	Normal usually.	Diminished.
Course:	Slow.	Rapid.
Loss of weight:	Gradual.	Rapid.
Cachexia:	None, or slow emaciation.	Rapid and peculiar type.
Tumor:	None.	Present later.

The possibility of achylia gastrica being the cause of *various types* of gastro-intestinal disturbances, of chiefly intestinal derangement or irregularities, or of severe anemia must be considered. Its association with pernicious anemia, gastric neuroses, various nervous symptoms, gastroptosis, and mucous colic must be remembered. These facts further emphasize the importance of gastric analysis.

Treatment.—This depends on the cause. Rose's belt, if achyli is due to gastroptosis or mucous colic, is necessary. If associate with nervous affections, these conditions should receive treatment Such patients should have their nutrition improved, and placed o the diet shortly to be laid down. As in these cases achylia is a functional disturbance, the secretion should be stimulated by—

Strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021), t. i. d. before meals, o condurango (fluidextract), ♏xv to xx (0.88–1.18), or tinct. nu vomica, ♏v to x (0.29–0.59), with the addition of hydrochloric acid thus:

℞.	Tinct. nucis vomicæ Acid. hydrochlor. dilut.	āā ʒiij (12.0)
	Comp. tinct. cinchona	℥j (30.0)
	Pure pepsin	ʒiss (6.0)
	Aq. destil. q. s. ad.	℥iv (125.0).—M.

Sig.—ʒij in water t. i. d. before meals (preferably).

Pancreatin, gr. v to x (0.3–0.6), with soda bicarb., gr. xv (1.0) or taka-diastase, gr. v (0.33), can be given after meals in cases wit diarrhea to aid digestion of starchy food.[1]

In the general treatment of achylia gastrica endeavor: (1) T stimulate gastric secretion. (2) To arrange the diet so that th food is easily digested.

To *stimulate gastric secretion* employ stomachics, such as nux vomica, condurango, etc., fifteen minutes before meals, as just described. The occasional use of lavage twice a week is of service in some cases. Litten advises the use of a 2 per cent. dilute hydrochloric acid solution for this purpose.

Intragastric faradization (preferably) twice a week or percutaneous faradization may be valuable. I would not advise this in the purely nervous cases.

If the symptoms simulate hyperchlorhydria, the use of water, ℥viij (250 cc.), or crackers and milk one to two hours after meals, as suggested by Einhorn, lessens the irritation.

Diet.—This is very important. Food should be prepared so as to pass readily from the stomach, and should be finely divided and thoroughly masticated. Meat should be given in small quantity, finely chopped, scraped (rare or broiled), chicken, pigeon, raw scraped beef, calves' brain, steak, fish, and game.

Starchy foods are excellent and should be thoroughly prepared; membranes covering any such should be removed. Starch is rapidly converted into sugar.

Mushes and liquids are preferable. Pea and bean soup (strained), barley, oatmeal, rice, sago, and potato soups. Mushes from rice. potato, tapioca, oats, flour, etc.; puddings. Soft-boiled eggs; yolks of eggs in soups; raw eggs beaten with water or milk; koumyss; matzoon; bacillac; bread; crackers; milk; plenty of cream and butter are indicated.

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[1] Cellasin tablets, gr. v (0.3) t. i. d., are also useful.

Somatose, sanatogen (plain or flavored), and meat powder; Valentine's or Liebig's beef-juice; Armour's extract of beef.

Cocoa possesses considerable nutritive power. Tea and coffee well diluted with milk can be given. My personal view is that alcoholic beverages are objectionable, though some allow them in moderation.

The second indication in the feeding is to employ it at frequent intervals, about every three hours, in small quantities, but so that the sum total shall be considerable. The object should be rather to overfeed and to increase the patient's weight by selecting those foods which best agree in each case.

In diarrheal cases, constipating food, such as potatoes and rice, should be selected. Feed by the "scales" is my rule in cases where loss of weight has occurred.

For the preparation of meat powder, either of the following methods is excellent:

(1) *Debove's Method.*—Roast finely chopped lean beef on a tinplate until desiccated, then powder in a mortar.

(2) *Einhorn's Method.*—Dry finely chopped meat three to six hours on a stove; then pound in a mortar and grind twice in a coffee-mill.

Butter, milk, cream, eggs (soft boiled or raw), potatoes, rice, peas, and beans (strained through a colander), cocoa, chocolate, and a small amount of beef, chicken, or game, and crackers or stale bread always seem to agree. I have often given 4 to 8 raw eggs a day. For example:

		Calories.
8 A.M.	Oatmeal with cream, 150 gm.	400
	Cocoa with milk, 200 gm.	135
	Soft-boiled eggs (2)	160
	Toast (2 slices)	160
	Butter, 20 gm.	163
11 A.M.	6 ounces (200 cc.) milk with raw egg	240
	Crackers, 2 oz. (60 gm.)	150
	Butter, 20 gm.	163
2 P.M.	Soup, bean or pea, 100 gm. (with 1 egg and 10 gm. of cane-sugar)	122
	Rare meat scraped, 100 gm.	215
	Mashed potatoes, 50 gm.	63
	Spinach, 50 gm.	30
	Bread, 2 slices (60 gm.)	135
	Butter, 20 gm.	163
	Tea and milk (2 lumps of sugar)	60
5.30 to 6 P.M.	Soft-boiled egg (1)	80
	Rice, 50 gm.; milk, 200 gm.	302
	Bread (1 slice)	67
	Butter, 20 gm.	81
	Tea and milk	60
9 to 9.30 P.M.	6 ounces (200 cc.) milk or koumyss	128
	Zwieback, 50 gm.	129
	Butter, 20 gm.	163
	Total calories	3269

It may be necessary to modify this diet and give less, especially at first.

In cases of severe anemia, rest in bed; arsenic in large doses, beginning with Fowler's solution ♏v (0.296) t. i. d. and increasing to ♏x to xv (0.59–0.88) t. i. d.; or by hypodermic in sterile water atoxyl, gr. ⅓ to ⅔ (0.022–0.044), every other day, and, in addition, peptomangan (Gude), iron tropon, or other good iron preparation.

Intestinal irrigation is of value in these cases when *putrefactive processes* are present in the intestine, for example, with ℥j to ij (30.0–60.0) peroxid of hydrogen or acetozone gr. xv (1.0) to the liter of water. Enteroclysis is especially advocated in pernicious anemia cases, with bacillac as diet.

I have recently found urotropin, gr. v to x (0.3–0.6), combined with equal quantities of benzoate of soda, is of great service as a remedy for intestinal putrefaction.

Resorcin and the other remedies suggested for this purpose under Chronic Gastritis and in the paragraph on Indicanuria are of value.

The bismuth preparations, notably bismuth subcarbonate or subnitrate, gr. x to xxx (0.6–2.0) t. i. d., can be given if diarrhea is present, and in this type boiled milk, potatoes (mashed), and rice are serviceable.

If there is dilatation of the stomach, lavage and the treatment for this condition must be carried out and Rose's belt applied.

CHAPTER XII

HEMATEMESIS—ULCER OF THE STOMACH—EXULCERATIO SIMPLEX—ACUTE AND CHRONIC EROSIONS

GASTRIC HEMORRHAGE

VOMITING of blood cannot be considered to be a proof of gastric hemorrhage, as it may come from the esophagus, nose, or mouth, or be coughed up and swallowed.

On the other hand, gastric hemorrhage may occur and the blood only appear in the stools.

The causes of gastric hemorrhage may be classified as follows:

1. Trauma over the stomach; injuries to the mucous membrane from foreign bodies, as bones or needles; damage from the stomach-tube, mineral acids, or caustic alkalis.

2. Thrombosis or embolism of the vessels; aneurism; varicosities; atheroma of a vessel, or fatty degeneration.

3. Venous stasis due to cirrhosis of the liver; tumors of the liver; pylephlebitis; compression of the vena cava.

In case of cirrhosis the hemorrhage is from the mucous membrane or from esophageal varices.

4. Lesions of the heart or lungs, causing stasis in the vena cava.

5. Constitutional diseases, as leukemia; pseudoleukemia; pernicious anemia; hemophilia; scurvy; purpura; melena (morbus maculosus neonatorum).

6. Menstrual type, when amenorrhea is present.

7. Lesions of the central nervous system (brain or spinal cord).

8. Hysteria.

9. Ulcer of the stomach and carcinoma.

10. Acute infectious diseases, as yellow fever, scarlet fever, measles, small-pox, etc.

11. Weil's disease (epidemic jaundice); malignant jaundice (acute yellow atrophy).

12. Phosphorus-poisoning.

13. Acute jaundice with hemorrhage, following operation.

14. Erosions—postoperative hematemesis due to these; the French describe them after appendicitis as vomito-negro-appendiculaire.

15. Exulceratio simplex (Dieulafoy), or superficial ulceration of the stomach.

As it is the general tendency to impute cases of gastric hemorrhage chiefly to ulcer or cancer, it seemed desirable to classify all causes.

Symptoms.—The chief symptoms are hematemesis and melena. Acute anemia develops if much blood is lost; the patient feels dizzy and weak and faints easily. The sight is blurred, pulse rapid an feeble, and extremities cold; rarely convulsions and death follow Nausea and vomiting occur. The blood may be dark in color o coffee ground in appearance, or light if in a large amount. A evanescent rise of temperature may occur after the hemorrhage.

At times the symptoms may take place with no hematemesi and nothing definite visible in the stool, when Weber's or the benzidi test may be necessary to determine the presence of blood.

Prognosis.—The prognosis is rarely fatal from the hemorrhag itself, but depends on the primary disease.

Treatment.—A hypodermic of morphin, gr. $\frac{1}{4}$ (0.016), and locall the ice-bag; ext. ergot ʒss (2.0) in solution by hypodermic, or ernuti ♏v (0.3) by hypodermic, and in addition gelatin 10 per cent. solution or Trémoliere's solution gelatin, 5 per cent. with calcium chlorid, per cent. These gelatin preparations should be given from ʒij to ℥ (8.0–30.0) every half-hour to an hour by mouth.

Tannic acid, gr. v (0.33), or lactate or chlorid calcium, gr. xv (1.0) should be given in solution (water 200 cc. or ℥vj) by rectum; lactat of strontium or magnesium, gr. xv to xxx (1.0–2.0) in 60 cc. (℥ij of water can also be administered by hypodermic.

Adrenalin chlorid (1 : 1000); 5 to 10 drops (0.291–0.582 cc.) by mouth or hypodermic is recommended, but it at times too rapidl increases pulse tension, especially if given hypodermically.

Hypodermoclysis or the rectal injection[1] of normal saline a 120° F. are useful. Ice-water lavage in rare instances may b necessary. Stimulants, such as strychnin, gr. $\frac{1}{60}$ (0.00108), o camphorated oil, gr. $7\frac{1}{2}$ (0.5), camphor in ♏20 (1.3) of sterile almon oil, by hypodermic, may be required.

ULCER OF THE STOMACH

(*Synonyms.*—Ulcus Ventriculi (Simplex); Peptic Ulcer; Ulcus Ventriculi Rotun dum; Perforating Gastric Ulcer; Cruveilhier's Disease.)

Ulcer of the stomach is characterized by a destruction of th mucous membrane of the stomach varying in degree, exhibiting n tendency to heal, and in typic cases attended with gastric symptom associated with pain, vomiting, and hematemesis. It was firs described by Cruveilhier in 1829.

Etiology.—*Postmortem and Geographic Distribution.*—Brinto found evidences of gastric ulcer in 5 per cent. of persons dying fro all causes, most frequent in London and on the Continent. Gerhard notes its frequent occurrence in Thuringia, and Von Sohlern it rarity in Russia, the Rhine region, and in the Bavarian Alps, believin this to be due to the vegetarian diet (rich in potassium salts) in thes countries. This theory lacks scientific confirmation.

Sex.—Gastric ulcer occurs more in women than in men. Welch places it at 60 per cent. in women and 40 per cent. in men, while Brinton believes it twice as frequent in women.

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[1] Proctoclysis is of value.

Age.—Cases have been reported in children under ten years. It occurs most frequently between twenty and forty in females, and in males quite often between forty and fifty. Ewald places the highest mortality between forty and sixty. It may occur in old people.

Occupation.—Cooks, shoemakers, and porcelain makers are most liable to this disease, but it seems a matter merely of coincidence.

Traumatism.—Simple trauma probably cannot produce gastric ulcer unless other conditions are associated. Blows, falls, and the swallowing of foreign bodies, such as knives by jugglers, etc., have produced severe damage to the mucosa of the stomach and marked hemorrhage, without the ultimate production of ulcer. Griffini and Vassale have resected or burned out portions of the mucosa of the stomachs of animals and perfect healing has taken place, with no ulceration. Traumatism may be a factor, as is shown in the following case of mine: Girl, age twenty-two, with no gastric symptoms, was thrown from a trolley car, striking on the epigastric region. Pain and tenderness were present at this point and gastric symptoms developed. There was no visible hemorrhage, but pain and symptoms continued for six weeks, apparently of hyperchlorhydria, but local tenderness persisted at the point of injury. The patient then had a sudden gastric hemorrhage of severe type and the ultimate course was of ulcer of the stomach. Cure resulted in eight months, the case being observed for several years subsequently, with no recurrence. This girl was anemic before the accident and probably hyperchlorhydria was present, though no symptoms were complained of. A hematoma was, I believe, produced in the stomach wall, interfering with its nutrition, and the other conditions favored ulcer development. The patient also had a hematoma of the thigh from the same accident. Traumatism under such conditions may be a factor.

Anemia and chlorosis may predispose to ulcer, and Riegel and Charles Stockton have shown that hyperchlorhydria frequently accompanies these conditions, and that it has an influence in the prevention of the cure of ulcer or even in its production. Experiments on animals have been performed for the purpose of studying the etiology of ulcer. Quincke and Daettwyler made animals anemic by venesection and produced lesions in the gastric mucosa. Section of the spinal cord, with the introduction of ½ of 1 per cent. hydrochloric acid solution in the stomach of a dog, has produced ulceration (Koch and Ewald). It has occurred after injury to the anterior corpora quadrigemina.

Weinland recently maintains that there is formed in the gastric mucosa an antibody, an antipepsin, which opposes the digestive action of the acid gastric juice. If the antibody is deficient in a certain area, this unprotected region is readily injured by the gastric juice. Rosenau has produced gastric ulcer by the injection of diphtheria toxin. Botton has produced gastric ulcer in animals by injecting the macerated gastric mucosa of other animals and of animals of other species.

The neurotrophic theory has been held by some. Stockton believes that nerve perturbation analogous to herpes may be a factor; and de la Verdora produced ulcer and hyperchlorhydria by injecting alcohol into the splanchnic and celiac plexus of a dog. Section of the vagi below the diaphragm has caused ulceration of the gastric mucosa. These experiments suggest that the nervous system may be a factor.

Silberman introduced substances into the blood producing hemolysis (hemoglobinemia) with resulting anemia, and found that defects of the mucosa healed tardily. Türck has produced gastric ulcer by feeding dogs with pure cultures of the bacterium coli commune. It is known that burns of the abdomen may produce gastric ulcer, though usually the duodenum is affected.

Pavy held the theory of diminished alkalinity of the blood, but this can hardly be accepted. Erosions have been considered by some to be the cause of ulcers, but Langerhans opposed this from his experience in autopsies, and Einhorn has demonstrated that gastric erosions are a clinical entity and that ulcer does not result. We know that autodigestion of the gastric mucosa occurs after death. The effect, therefore, of circulatory disturbances of the blood-vessels of the stomach in the development of ulceration is important.

Virchow first suggested that ulceration may result from the plugging of a nutrient artery to part of the mucosa by a thrombus or embolus, and that the infarction is destroyed by the gastric juice. Panum supported this view by producing infarcts and ulcers of the stomach in a dog by injecting an emulsion of wax into the femoral artery. Occlusion of the portal vein or of some of the large veins of the stomach may cause gastric ulcer.

Injection of chromate of lead into the gastric and splenic arteries (Cohnheim) has produced ulcer. Talma, by increasing the tension of the gastric wall by ligating the orifices of the stomach, has brought about ulceration. Artificial anemia by faradization of the stomach has caused ulcer. *Local interference with the circulation*, with *resulting necrosis*, associated with hypochlorhydria and changes in the blood are probably the most frequent causes.

Hyperchlorhydria is present in about 95 per cent. of the cases,[1] but occasionally there is subacidity or achylia gastrica, as reported by Einhorn and others. I have recently seen a case with very low acidity (Connor's case).[2] Probably gastric ulcer is not always produced by the same factors, and several of the theories described may apply. Hyperchlorhydria undoubtedly has a bearing on most cases, and frequently anemia or chlorosis.

Morbid Anatomy.—The peptic ulcer is found in the regions exposed to the gastric juice, in the stomach, lowest part of the esophagus, and upper duodenum. It is round or oval, occasionally oblong; is funnel shaped, the upper part being the larger; is of variable depth, its floor being formed by the submucosa, muscular tissue, serosa, or by adjacent adherent organs. The acute ulcer is usually small, punched out, with clean cut edges and a smooth floor, with no thickening of the peritoneal coat; occasionally the floor may be covered with a thick green or brown mucus.

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[1] Recent reports from Mayo's cases show there are quite a number without marked increase in HCl.

[2] Frequent gastric analyses showed free HCl trace or absent; lactic acid present; *microscopic pus* and occult blood. Multiple ulcers (non-malignant) were found by John Connor at operation.

The chronic ulcer is of larger size, with callous margins, and the border may be sinuous. It is often markedly indurated, so if situated at the pylorus it may feel like a tumor on palpation.

Embolism or endarteritis of the artery supplying the ulcerated region has been found, or a small aneurism on the floor of the ulcer.

Microscopically.—The ducts of the glands are cut off toward the base, being eaten away or digested up to where the tissue offers sufficient resistant power to the gastric juice.

Healing occurs by proliferation of the connective and glandular tissue near the margin of the gland. As the connective tissue contracts, the proliferation of the glands is stopped. If the stomach wall is adherent to an adjacent organ and the ulcer perforates, the neighboring tissue may grow into the hole and unite with the connective tissue growing from within. Muscle-fibers do not regenerate. This is true of the large deep ulcers; the mucosa and muscularis roll in and adhere to the adjacent organ. There is further proliferation of tissue caused by irritation of the gastric juice, and the latter may cause erosion of vessels.

The rest of the *mucous membrane of the stomach*, as a rule, *remains normal.*

Extent of the Ulcer.—It may vary from the size of a pea to a diameter of 5 or 6 inches; the average being from a 5-cent piece to a 25-cent piece. Peabody reports one measuring 19 by 10 cm.

Location.—It is commonly situated on the posterior wall of the pyloric end of the stomach, at or near the lesser curvature. Welch states that out of 793 cases, 288 were in the lesser curvature, 95 at the pylorus, 96 on the anterior wall, 50 at the cardia, 29 at the fundus, and 27 on the greater curvature. Other statistics are given.

Number.—In about 80 per cent. of cases 1 ulcer is found; in a trifle over one-half the remainder, 2 ulcers; in the balance, 3 to 5 ulcers; Osler reports 34 ulcers in 1 case, and Lange 1 in which he could not count them.

Progress of the Ulcer.—1. Cicatrization may occur, with formation of a connective-tissue scar, which tends to depress and contract. Depending on its location, it may cause stricture of the pylorus, esophagus, or an hour-glass stomach. In other situations it may produce no trouble.

2. Progressive necrosis may take place and there may result:

(*a*) Erosion of a blood-vessel with severe or occasionally fatal hemorrhage from perforation of a large vessel, such as the gastric, hepatic, or splenic artery, portal vein, etc.

(*b*) Adhesions to neighboring organs or various perforations. The stomach may become adherent to the liver, gall-bladder, spleen, pancreas, or intestines, and there may be perforation into these organs.

If the ulcer is on the anterior surface, then direct perforation and general peritonitis may follow.

There is sometimes a circumscribed peritonitis when adhesions form with other organs and a local abscess, which may later perforate into the peritoneal cavity.

The ulcer may perforate into the lesser peritoneal cavity and cause subphrenic abscess; the diaphragm may be perforated and a communication formed with the pleura, lungs, pericardium, and even with the left ventricle; or, rarely, an emphysema of the subperitoneal tissue occurs, which may pass into the posterior mediastinum; or adhesions may form and a perforation of the anterior abdominal wall take place; or general emphysema of the subcutaneous tissues may rarely result.

In cases with adhesions to adjacent organs, with or without abscess, localized growing tumors, hard in consistency, may be formed, and Gerhardt notes the possibility of mistaking these for carcinoma, but the history and gastric analysis make the diagnosis.

Symptoms.—Some cases present typic symptoms, in which event the diagnosis is easy; others suffer apparently from a simple hyperchlorhydria for a long period of time with no special symptoms pointing to ulcer; while in others the condition is latent.

In the *latent cases* the patient is sometimes apparently perfectly well, when there will suddenly develop hematemesis or symptoms of perforative peritonitis. Others may not vomit, but suddenly turn faint and weak, become pale with a feeble pulse, presenting the *symptoms of internal hemorrhage.* Examination of the stool for occult (concealed) hemorrhage by Weber's, the aloin, or the benzidin test is a valuable aid.

Cases Simulating Hyperchlorhydria.—Kaufmann,[1] of New York, has suggested that in cases of hyperchlorhydria not yielding to treatment and in whom the pain is of a gnawing or tearing character the suspicion of ulcer is justified. This is undoubtedly true, and repeated examinations of the gastric contents and stool for blood or occult blood are indicated.

Typical Case.—As a rule, the symptoms of gastric ulcer develop slowly and are as follows: at first a feeling of fulness and pressure after eating; gradually increasing to pain in the epigastrium, which may become so severe that the patient is afraid to eat. Nausea, regurgitation, or vomiting may occur early.

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[1] New York Medical Journal and Philadelphia Medical Journal, March 11, 1905.

Pain occurs *generally a few minutes after eating*, though sometimes one-half to one hour later, and persisting during digestion. Coarse substances and large quantities of food increase it. It is of a burning or gnawing character. Epigastric pain is increased on pressure and the sensitive point is usually circumscribed.

A few weeks later dorsal pain begins, gnawing in character, lying to the left of the spine, between the eighth and tenth vertebræ, alternating with the epigastric pain; at times there is sensitiveness on pressure in this region.

The epigastric pain is not continuous, as in cancer, but there are periods of relief. Later, vomiting may occur, one to two hours after meals, of very acid watery material, mixed with food; *emesis generally relieves the pain;* occasionally there is vomiting late at night or early in the morning if gastrosuccorrhea (hypersecretion) is associated with ulcer.

Appetite is variable; at times the patient desires food, but fears to eat on account of pain; constipation generally marked; amenorrhea frequent in women; anemia marked in many cases.

These symptoms continue, then hemorrhage suddenly occurs, and is visibly present in one-third to one-half the cases, and in a very large percentage *when occult hemorrhage* is included, as it should be.

(1) *Hemorrhage may be occult*, no vomiting of blood, the patient turning pale and faint and in a cold sweat, and on the next day there are tarry stools, or occult blood is found in the stool by Weber's or the benzidin test. Progressive anemia may be caused by small repeated hemorrhages.

(2) The patient may experience a sense of fulness after a meal and become nauseated and restless; then hematemesis occurs in large amount, of fluid blood, bright red, or of liver color, brown, or coffee grounds, mixed with food.

Patient may feel faint, extremities cold, temperature subnormal, become collapsed and, rarely, even die from hemorrhage. Convulsions and unconsciousness may precede death. Death from internal hemorrhage can occur without vomiting. Blood is generally passed by the stools (melena), black and tarry in color, and may be found in cases with no vomiting or in latent cases.

Convulsions from cerebral anemia, or hemiplegia from thrombosis, or amaurosis (possibly permanent) have occurred. A temporary rise of temperature may follow the collapse. Persistent temperature shows complications. The vomitus usually shows hyperchlorhydria and no mucus. Tongue is clean and red, rarely coated.

Often there are remissions and exacerbations of the symptoms, and they may be protracted; from no apparent cause a relapse may occur. With an unhealed ulcer, complications or perforation may occur at any time. In others, ulcer symptoms may disappear, but those of gastric dilatation, adhesions, etc., may follow. In the long cases, from pain and self-starvation, marked emaciation takes place;

the suffering shows in the patient's face, but there is not the sallow appearance of cancer. Tetanic attacks complicating chronic ulcer have been reported by Kaufmann.[1]

Pain.—The epigastric pain usually occurs about the center of this region, in the median line, just below the tip of the ensiform. Occasionally it is more to the right or left, and lies in a circular area of 1 to 2 inches in diameter. Throbbing and pulsation may be felt in the epigastrium. Pain is *usually increased on pressure,* rarely lessened. It is injurious to frequently manipulate the *painful area* or subject it to *marked pressure.* Testing with the algesimeter is not advisable.

In place of vomiting, some patients regurgitate acid chyme, with pyrosis, or suffer from nausea.

Motor Function.—In uncomplicated cases of gastric ulcer this is increased. If pyloric stenosis or adhesions complicate, then the function is interfered with.

Examination of the Stomach Contents.—If the diagnosis of ulcer has been positively determined, it is preferable not to pass the tube. If there is no hematemesis, but vomiting is excessive, it is well to give the test meal and examine, in addition, for occult blood. Lavage after the contents are removed is of service, as it checks emesis, and thus renders a hemorrhage less liable.

If the patient states there has been a hemorrhage, but the diagnosis is in doubt, the tube should not be passed earlier than ten days to two weeks after the history of hematemesis. The stool can be examined at once for occult blood.

If vomiting occurs, the vomitus may be examined by preference, but the quantity and quality of the food and time of ingestion would influence the analytic findings and might lead to error. Immediate examination of the *vomitus and stool for occult blood is important.*

In about 95 per cent. of uncomplicated cases of ulcer, hyperchlorhydria is present; the total acidity is high, from 90+ to 150+, and free hydrochloric acid 50+ to 60+ or even 90+, and *there is no mucus.*

There are cases of subacidity, or even of achylia gastrica, with ulcer.

The Absence of Hyperchlorhydria Does Not Exclude the Presence of Ulcer.—In all *doubtful cases* gastric analysis and examination *for occult blood* in the *stomach contents and stool* should be carried out.

In some cases we must think of the possibility of development of carcinoma on the base of an ulcer, but in such event tumor growth and increasing cachexia occur.

Urine.—The quantity is reduced when there is much vomiting and food is diminished. There are no characteristic changes.

Complications.—Perforation and general peritonitis; circumscribed peritonitis; sacculated abscess; adhesions with other viscera, with or without perforation of them; pyloric stenosis; stenosis of the cardia; perigastritis with adhesions; subphrenic abscess; perforation of the diaphragm, pleura, lungs, pericardium or heart; hour-glass contraction of the stomach; external fistulous opening; anemia of severe type; mediastinal emphysema, and subcutaneous emphysema may occur.

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[1] American Journal of the Medical Sciences, April, 1904.

Stenosis of the pylorus, due to stricture or hypertrophy from spasm, produce dilatation of the stomach and its symptoms; stenosis of the cardia causes dysphagia and regurgitation of food.

Perforation.—This occurs in ulcers on the anterior stomach wall, or from perforation of a circumscribed abscess, with resulting general peritonitis. The symptoms are sudden pain, at times with a tearing sensation, shock, muscular rigidity, rapid distention of the abdomen; tenderness on pressure; disappearance of liver dulness; cold sweat; rapid and feeble pulse, followed by a rise of temperature, singultus; frequently vomiting; anxious and sunken face (facies Hippocratica); usually coma; then death. Leukocytosis, especially increase in the polynuclears, is present.

In perforation, with circumscribed abscess formation, the symptoms are less intense and are localized. Perforation of the stomach occurs usually after a full meal, or following coughing, sneezing, or local mechanic violence.

If adhesions form with other organs, these may be perforated.

Frequency of Perforation.—Brinton gives the frequency of perforation as 1 in 8 cases. Others place it at 6 to 7 per cent.

In women, Brinton places one-half the perforations at the age of fourteen to thirty, the average being twenty-seven; while in men it is distributed up to fifty, the average age being forty-two.

The chances of perforation of an ulcer on the anterior stomach wall are 5 to 1 in its favor, on account of its mobility which prevents adhesion formation, but ulcers are much less frequent in this location.

Subphrenic abscess (pyopneumothorax subphrenicus, when gas is present).

Etiology.—The chief causes are as follows: Posterior perforating ulcer of the stomach (the most frequent cause); traumatism of the liver; abscess of the liver; retroverted appendix; and perforation of duodenal ulcer.

The boundaries of the abscess-cavity are: above, the diaphragm; below, the stomach and liver; to the left, the spleen; to the right, the suspensory ligaments of the liver. The liver is pushed down and the diaphragm upward.

Symptoms.—These are abrupt when due to perforation of gastric ulcer and are as follows: severe pain; vomiting of bilious or bloody material; embarrassment of respiration; subsequently chills, fever, and emaciation. Leukocytosis and increased polynuclears during the suppurative period.

Physical signs depend on the quantity of air in the cavity and upon the presence or absence of a complicating pleurisy.

Physical Signs (with Little Air Present).—Dulness or flatness in the lower part of the thorax, but cough and expectoration are absent; signs of pressure in the pleural cavity are absent or slight, the thorax not being much dilated, and there being scarcely any obliteration of the intercostal spaces; the lungs are intact and distensible, and on deep inspiration there is vesicular breathing.

Physical Signs (with Much Air Present).—The lower part of the thorax protrudes and respiratory movements diminish; the heart is sometimes pushed upward and slightly to the right; the *liver extends well down into the abdomen*, occasionally as far as the umbilicus; the liver dulness in the back and lower part of the lung is replaced by a tympanitic zone; on auscultation the respiratory sounds are absent in this zone, and there are succussion sounds of a metallic pitch.

When pleurisy complicates subphrenic abscess from ulcer, there are the signs of pleurisy. Senator gives the following diagnostic points:

Violent pain in the epigastric and hypochondriac region; pain in the back on sitting up; pain on belching; patients prefer dorsal position when abscess complicates; while in pleurisy alone, they lie on the diseased side; edema of the lower lateral and posterior thoracic walls.

Pfuhl suggests a diagnostic point between *subphrenic abscess* and *pyopneumothorax*.

In subphrenic abscess, if an aspirating needle be inserted and a manometer be attached, the pressure is greater on inspiration and less on expiration.

In pyopneumothorax the pressure conditions are reversed.

Exploratory puncture is the accurate method of diagnosis, pus and food particles being aspirated.

Terminations of Subphrenic Abscess.—Perforation of the diaphragm and pleura; perforation of the lung, with expectoration of pus; or of the pericardium; or of the left ventricle; or of the colon; rarely perforation into the general peritoneum; or perforation of the skin, with resulting fistula.

Successful operations have been performed for subdiaphragmatic abscess, notably by Carl Beck.[1] Tuberculosis may occur in association with ulcer.

Some authors describe so-called atypic forms of ulcer, taking the most prominent symptom as a basis, such as: Gastralgic. Catarrhal or vomiting. Dyspeptic or latent. Hemorrhagic. Cachectic. Perforative form.

Diagnosis.—This is easy in the typic form characterized by hematemesis, the epigastric pain circumscribed, and present during the digestive process, the dorsal pain, local tenderness, and vomiting.

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[1] Medical Record, Feb. 5, 1896.

DIFFERENTIAL DIAGNOSIS

	Ulcer of stomach.	Nervous gastralgia.	Cancer.	Hyperchlorhydria.	Cholelithiasis.
Age.	Chiefly twenty to forty to fifty.	Eighteen to forty years.	Middle age and advanced life.	All periods, except in youth.	Over forty mostly; rare under twenty-five.
Sex.	More in women.	More in women.	More in men.	More in men.	Mostly in women.
Epigastric pain.	Intense; appears shortly after meals; increases on pressure; disappears at end of digestion; some free periods during the day at times, if stomach is empty.	Irregular; does not depend on food; relieved by pressure; duration of several days free period from pain. Disappearance of pain after electricity (Leube).	Pain less intense, but continuous; seldom free period, and is more steady; local tenderness.	Pain one to three hours after meals, and disappears after albuminous food or soda bicarbonate are given.	Paroxysms of pain independent of eating often late and in the right hypochondrium. Pain over gall-bladder, which at times is enlarged and painful. Alkalis and albuminous food do not relieve pain.
Dorsal pain.	Present.	Absent.	Absent.	Absent.	Head's gall-bladder zone.
Appetite.	Not impaired; afraid to eat.	Variable, peculiar cravings.	Poor.	Often increased.	Pain attacks patient in perfect health and radiates to right side or to right shoulder. It may cause collapse on account of severity.
Tongue.	Dry, red, and clean; or moist and smooth, with light fur.	Normal.	Coated thickly.	Clean or slight fur.	Temperature and chills may occur. Liver may be enlarged and sensitive. Icterus occasionally.
Taste.	No abnormality.	No abnormality.	Bitter or sour.	No abnormality.	Gall-stones in the stool.
Belching.	Absent usually; no odor if present.	Some.	Present, with disagreeable or fetid odor.	Absent.	Vomiting may be present. No dorsal pain. Hyperchlorhydria may be associated, but the history helps differentiation. Leukocytosis and increase in the polynuclears are present if there is inflammation, and aid in diagnosis.

DIFFERENTIAL DIAGNOSIS

	Ulcer.	Nervous gastralgia.	Cancer.	Hyperchlorhydria.
Regurgitation.	Present at times; waterbrash with pyrosis.	Rare.	No waterbrash; pyrosis present.	Waterbrash and pyrosis.
Vomiting.	Soon after meals, during digestion.	No regularity; occasional.	Once or twice a day, or every day or so, and not after each meal; quantity large.	None, unless hypersecretion be associated.
Hematemesis.	Vomiting of often large amount; red or coffee ground; blood in stool (melena); often repeated same day or next day and then none for some time. Occult blood.	None.	Vomiting of blood, generally in small quantities; color usually of coffee grounds. May occur at frequent intervals. Is often decomposed and fetid in odor; melena.	None.
Tumor.	None; unless thickened ulcer at pylorus, which then is smooth.	None.	Palpable; uneven; hard, tender, and readily movable.	None.
Secretory function.	Increased, as a rule. HCl increased usually. Lactic acid absent.	Variable. No lactic acid.	HCl diminished or absent usually. Lactic acid present.	HCl increased. No lactic acid.
Perforation.	May take place early.	Absent.	Rare; in last stages.	Absent.
Complexion.	Often fresh; may be anemic after losses of blood; face anxious and suffering in old cases. Some loss of weight in old cases, but more gradual.	Pale.	Sallow and yellow; skin dry; peculiar and marked cachexia; rapid loss of weight. Leukocytosis is moderate; eosinophilia.	Often pale.

Other conditions have been mistaken for gastric ulcer. *Gastric crises of locomotor ataxia;* absence of patellar reflexes; Rhomberg symptom and the Argyll-Robertson pupil are diagnostic.

Duodenal Ulcer.—This is at times impossible to differentiate, especially if the ulcer is near the pylorus. It occurs most frequently *in males;* is often latent; melena is frequent; pain and tenderness are often a little more to the right than in gastric ulcer; no dorsal pain; hematemesis not as frequent; pain after the ingestion of food is usually later than in gastric ulcer. Occult blood most frequent in stool.

Spider Gall-bladder Adhesions.—Robert T. Morris has demonstrated that gastric hemorrhage occurs at times with this condition. The stomach is dilated, and the diagnosis has been made of stenosis of the pylorus with ulcer.

There is a previous history of gall-bladder disease in these cases. The possibility of this condition must be considered.

Cirrhosis of the Liver.—Severe gastric hemorrhage may occur, but examination of the liver, the history, and other symptoms will differentiate.

Location of the Ulcer.—Occasionally one can make a probable diagnosis as to the position of the ulcer; if relief is afforded when standing, the ulcer is probably on the lesser curvature; if pain is intense on standing, it is on the greater curvature; if less severe pain when lying on left side, the ulcer is probably at the pylorus, etc. The position which affords most comfort to the patient is the *one which permits the ulcer to remain above the gastric contents.*

Einhorn[1] has recently described two methods for recognizing and localizing gastric ulcers: the so-called "thread impregnation test," in which the patient swallows the duodenal bucket with thread attachment, which is removed twelve hours later. Blood discoloration on the string shows the presence of ulcer, and the distance of this spot from the teeth, its location. His second method, by the "gastric stamper," consists in the introduction of a collapsed balloon into the stomach, which is then distended and secures an impression of blood from the ulcer. It is then deflated and removed. Both methods are uncertain, and pressure from inflation, I believe, dangerous. The use of the gastroscope to locate the ulcer is a risky procedure. The presence of blood or occult blood in the gastric contents, vomitus, or stool, together with the methods of diagnosis previously described, are sufficient.

Course.—Gastric ulcer occasionally runs a rapid course with death from perforation or hemorrhage. Stowell[2] states that 18 per cent. last a year or less; 46.5 per cent., from one to six years. A case of thirty years' duration has been reported. There are often intermissions of improvement and exacerbations; or the patient may become a chronic invalid.

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[1] Medical Record, April 3, 1909.

[2] Ibid., July 8, 1905.

Prognosis.—Excepting the fulminating cases, the more recent the ulcer, the more favorable the prognosis as to cure.

The mortality has been estimated at from 8 to 10 per cent.; some place it up to 20 per cent. The position of the ulcer modifies the prognosis; if on the anterior wall, perforation is more apt to occur; if the pylorus is involved, stenosis and dilatation of the stomach result; if hypersecretion be associated, the results are less favorable. Stowell's statistics are as follows:

Death from hemorrhage, 3 to 4 per cent.; from exhaustion, 5 per cent.; from fatal perforation, 6.5 to 13 per cent. Pulmonary tuberculosis was the terminal event in 20 per cent. (Debove and Rémond) out of 100 cases investigated.

Treatment.—*For Hemorrhage (Hematemesis).*—Absolute rest in the dorsal position; immediate injection of morphin sulphate, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), and the application of a light ice-bag over the stomach, if necessary, suspended from a barrel hoop to avoid weight. In emergency I have tied bits of ice in a sheet of rubber tissue or in dress shields.

One to two teaspoonfuls of a 5 to 10 per cent. solution of gelatin (cold), depending on the severity of the case, should be given by mouth every half-hour for ten to twelve hours, even if vomiting. This is an excellent hemostatic, and also takes up the free acid.

The frequent administration of small quantities is preferable to larger amounts given every two or three hours.

Gelatin, 100 cc. (℥iij) of a 2 per cent. solution by hypodermic with a large syringe, given between the lowest rib and crest of the ileum, is of value.

Ernutin (Borrows and Welcome), ♏v to x (0.33–0.66), by hypodermic, or,

℞. Ext. ergot .... gr. xv (1.0)
Glycerini }
Aq. destil. } .... āā ʒj (4.0)

by a large syringe subcutaneously have proved useful adjuncts; or fluidextract of ergot or hydrastis, ʒj to ij (4.0–8.0), in 2 ounces of water by rectum.

Chlorid of iron and acetate of lead are objectionable.

Tremolière has recently advocated a solution as a local styptic consisting of a 5 per cent. gelatin solution containing 2 per cent. chlorid of calcium. In a severe case I should not hesitate to give at once by mouth 1 to 2 ounces of a 10 per cent. gelatin solution containing 5 to 10 gr. (0.3 to 0.6) of chlorid or, preferably, lactate of calcium.

The drug is dissolved in a little water and added to the gelatin solution while still warm and then rapidly cooled off on ice.

The plain gelatin solution should be administered *in every case.*

Lactate, or chlorid of calcium, gr. x to xv (0.66–1.0), in ℥iv (125 cc.) warm water can be given by enema as an adjuvant.

Lactate of strontium, or lactate of magnesium, gr. xv to xxx (1.0–2.0), in ℥ij to iij (125 cc. to 200 cc.) of sterile water, are recommended by hypodermoclysis, by Maas, to increase the coagulability of the blood, and would be valuable in gastric hemorrhage. Calcium chlorid should never be so given, as it causes local coagulation necrosis, and the lactate of calcium never stronger than 1:20 by hypodermic, and even so there is danger.

Adrenalin chlorid, 1:1000, ♏x to xv (0.66–1.0), have been recommended by hypodermic for internal hemorrhage, but the pulse tension is markedly increased thereby and I have seen secondary hemorrhage result. Five drops may be given by the mouth in ʒij (8.0) of water for the local styptic effect if other remedies are insufficient.

Bismuth subnitrate, ʒss (2.0), in 1 ounce of water is at times of service.

Ewald recommends cautious lavage with ice-water after cocainizing the pharynx (2 per cent. solution of cocain is sufficiently strong by spray), in cases in which death seemed imminent from continuous hemorrhage. I have never found the procedure necessary, though I have treated severe cases. If it is employed, the addition of 125 cc. (℥iv) of a 5 per cent. gelatin solution, gr. xv (1.0) lactate of calcium, and ♏x (0.66) adrenalin to the water for lavage would be of advantage.

Wiel advises lavage with hot water at 42° C., but I doubt its advisability. When all methods fail and the hemorrhage continues, opening the stomach and direct suture of the bleeding ulcer has been advocated.

For Thirst.—The cold gelatin given for hemorrhage helps relieve thirst. A small piece of gauze dipped in cold water and held in the mouth, frequent washing of the latter; an occasional pellet of ice and enemata of normal saline solution at 105° F., ℥iv to vj (125 cc. to 200 cc.), if no relief by other methods, and given every three hours.

Proctoclysis, the administration of saline solution per rectum by the drop method, is also useful.

Collapse.—Caution must be employed not to overstimulate the heart, lest the coagulum be forced out and hemorrhage recur.

Only collapse that is fairly marked—a pulse 120 or over—should be treated, as depression of the pulse favors clotting. Among the valuable methods are:

External application of heat to the limbs by hot-water bags; rectal injections of 1 pint (500 cc.) to 1 quart (liter) of hot normal salt solution at 115° to 120° F. every two to three hours, also proctoclysis can be employed.

Hypodermoclysis with normal saline solution, ℥vj (375 cc.) to Oj (500 cc.), given at 105° F., through a large needle attached to a fountain syringe, preferably between the iliac crest and twelfth rib, is valuable and does not stimulate too rapidly.

In emergency I saved one patient by employing an ordinar hypodermic needle and elevating the fountain syringe 6 to 8 fee to obtain forcible flow. In this event a higher temperature (118° F. should be employed, as the fluid cools in passing through the smal needle.

In one case, having no time to perform infusion, I needled a larg superficial vein with a hypodermic needle[1] and infused by this method Infusion with 1 liter normal saline solution at 120° F. in sever cases is indicated. Direct infusion of blood from donor to patien is generally impractical and the simple method is the best.

Strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021), every three or fou hours by hypodermic or camphor. pulv., gr. v (0.3), in sterile almon oil, ♏xx (1.184 cc.).

A single dose by hypodermic and repeated in three or four hours if necessary. Bandaging the extremities and elevation of the foot of the bed are of service.

Whisky or brandy, ℥j to ij (30.0–60.0), may be added to the enema.

The rational methods for the cure of acute or chronic gastric ulcer differ widely, the chief exponents being Leube-Ziemssen and Lenhartz.

General Principles.—There are certain *general principles* to which I must first refer.

Absolute rest in bed for a period of two to four weeks, even though the hemorrhage may not have taken place recently, gives the best results.

Cruveilhier first recommended milk in gastric ulcer as an ideal food. Pure milk coagulates rapidly with some patients and does not agree. Boiled milk does not form large curds and leaves the stomach more rapidly. Lime-water or milk of magnesia lessens milk coagulability.

Lenhartz believes that the general nutrition should be improved as rapidly as possible to hasten the healing of the ulcer, and that sufficient milk to secure this result requires so large a quantity that it would overdistend the stomach. Though given in divided doses, he states that 2 to 3 quarts per diem are excessive, and limits the total quantity of milk to 1 liter, adding other materials to increase the calorie value.

Riegel demonstrated the capacity of egg-albumen to bind free hydrochloric acid, and also the use of sugar solutions to lessen acid secretion, such as pure dextrose or even ordinary dextrose. These possess high calorie value.

Soluble dry peptonoids—℥j (30.0)—have a calorie value of 120.4. They dissolve in milk or water. Milk powder (gm. 100); Gartner's fat milk; casein nutrose, gm. 30 to 60; somatose, ʒiv to vj (16.0–24.0), and notably sanatogen, 20 to 30 gm. per diem, possess nutritive qualities.

Among other foods are meat jelly, prepared by boiling chicken or beef with calves' feet (Fleming), Leube-Rosenthal's meat solution; flour soup boiled with milk; and barley-water or rice-water to dilute the milk.

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[1] Manual on Enteroclysis, Hypodermoclysis, and Infusion (Kemp).

Protection of the gastric mucosa and lessening hyperchlorhydria by the use of olive oil—℥ss to j (16.0–32.0)—t. i. d. before meals is of service.

Kaufmann[1] has demonstrated that the normal gastric mucus exercises a *protective influence* and is absent in ulcer, and that silver nitrate both aids the healing of ulcer and stimulates mucous secretion. Türck substantiates this by showing the increase of mycogen cells during the process of healing.

Large doses of bismuth act as a protective layer to the ulcer and prevent irritation.

Neutralization of the free hydrochloric acid is important. Among the most valuable alkalis are magnesia usta, milk of magnesia (Phillips), and soda bicarbonate. Magnesia preparations are also of use for the constipation. If their action becomes excessive, bismuth can be combined.

At times I employ soda bicarbonate in combination with magnesia. Bicarbonate alone is apt to produce too much carbonic acid gas and distend the stomach.

In cases without dilatation, Carlsbad water or the salts are of service, as they lessen gastric secretion and empty the bowels. They form a permanent feature in Leube's cure.

Belladonna is valuable when there is hypersecretion, gr. ⅓ (0.022) of the extract or ♏10 (0.6) of the tincture three times a day.

Iron and arsenic are imperative to improve the condition of the blood. Alcoholic drinks, coffee and tobacco, heavy salads, hot breads, acids, pastry, etc., should be excluded during the course of treatment.

If nutritive enemata are given for twenty-four hours following Leube's method, plain milk should never be employed, but should always be *peptonized*. This feature is often overlooked. An enema of saline solution should be administered an hour or more before the nutritive enema in order to cleanse the bowel. The following is of service: Peptonized milk, 125 cc. (℥iv); raw egg, or whites of 2 eggs beaten up; salt, 1.00 (gr. 15); sanatogen, 8.0 (ʒij); water, q. s. 250 cc. (℥viij).

Somatose, ʒij (8.0), or dry peptonoids, ʒj (4.0), or liquid peptonoids, ℥j (32.0), may be substituted for sanatogen. Four nutritive enemata should be given during the day of sixteen hours.

Metzger has shown that wine in the enema increases gastric secretion.

Having enunciated the general principles of treatment, I will describe the chief methods employed and the procedures with which I have been most successful in my own experience.

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[1] American Journal of the Medical Sciences, Feb., 1908.

Riegel confines himself exclusively to rectal feeding for six or eight days at the commencement of treatment following hematemesis, giving only a few pieces of ice by mouth, and then follows with a mild, non-irritating diet and the use of Carlsbad water or salts; while others confine the feeding to the rectum for two to three weeks.

Good results have been reported, but as the patient is suffering from subnutrition, these methods can be improved upon.

Leube-Ziemssen Rest Cure.—This is substantially as follows: The patient is kept in bed for two to three weeks, not being allowed to rise for any purpose, either for defecation or urination; rectal feeding *for several days* if hematemesis has just occurred; hot poultices (flaxseed) over the stomach by day and warm Priessnitz compresses at night. Then after the first two to three days the subsequent diet for ten days should consist chiefly of milk, or milk with barley-water, strained barley, oatmeal- or rice-water, tea and a little bouillon. For the next ten days boiled calves' brain, boiled thymus, rice and sago in milk, gruels and mushes, raw and soft-boiled eggs. This is followed by a little scraped rare or raw beef. Scraped raw ham and mashed potato for a week or so are added, and finally broiled chicken, venison or roast beef, pike and shad, etc.; coarse bread, skin, tendons, fruits, alcohol, and acids should be avoided.

Carlsbad water, a glass, or ʒj to ij (4.0–8.0) Carlsbad salts in ℥viij (250 cc.) of water should be taken half an hour before breakfast. This can be begun after the first week or ten days.

Einhorn[1] employs nutritive enemata for a day or so after hematemesis, and then milk as the basis of his diet for the first two weeks; for the first week giving ℥v (150 cc.) every hour, adding barley-water and bouillon; and gradually increasing the quantities of milk, but giving it at longer periods, adding eggs, crackers, etc., at the end of ten days, gradually increasing the diet.

Lenhartz's Method.—Lenhartz[2] believes that many cases of gastric ulcer do not definitely improve, or but very slowly, under the method of entrenched milk feeding; that the high acidity is not measurably lessened; and that if patients are in a poor physical condition consequent upon one or more hemorrhages, often, indeed, in collapse, the "starvation treatment"—the ice and nutrient enemata and insufficient milk feeding following—not only maintain the patient in his anemic state, but may even drag him into serious inanition, and such an undermined constitution hardly favors the speedy healing of an ulcer. Frequent nutrient enemata excite the gastro-intestinal tract into peristaltic activity and may thus induce renewed bleeding; besides, very little nutriment is, after all, obtainable.

Should more milk be given by mouth, merely enough to preserve the body weight—3 liters for an adult—would overfill the stomach and stretch its walls, thus preventing a contraction of the ulcer and again offering the danger of renewed bleeding. He advises another dietary treatment, one that will especially combat the hyperchlorhydria and reinforce the enfeebled and anemic state of the patient.

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[1] New York Medical Journal, Nov. 20, 1909. This author now employs raw eggs and milk from the first day.

[2] International Congress at Wiesbaden, 1901; Therapeutic Gazette, Nov. 16, 1906.

The concentrated egg-albumen diet was tried. In case after case the effect proved so gratifying that this method became the routine treatment. The sour regurgitation subsides, the vomiting immediately ceases, the pain and distress after eating, within a few hours to a few days, disappear, and finally an increase in the body weight is manifest as early as the first week. Besides, the improvement is comparatively rapid, so that the patient can be dismissed as cured within a briefer time than formerly.

The following is the tabulated regimen: "Absolute rest in bed for at least four weeks. All mental excitement to be avoided. An ice-bag is placed upon the stomach and kept there almost continually for two weeks. This prevents gaseous distention and promotes contraction of the stomach walls, thus tending to obviate hemorrhage, and eases the pain when present. On the first day, even when a hematemesis has occurred, the patient receives between 200 and 300 cc. (℥vj–ix) of iced milk given in spoonfuls and from 2 to 4 beaten raw eggs within the first twenty-four hours. At the same time bismuth subnitrate is given twice or thrice a day, 2 grams (gr. 30) per dose, and

	Days after last hematemesis.													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Eggs[1]	2	3	4	5	6	7	8	8	8	8	8	8	8	8
Sugar with eggs (gm.)	0	0	20	20	30	30	40	40	50	50	50	50	50	50
Milk (cc.)	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000	1000
Raw chopped meat (gm.)	0	0	0	0	0	35	70	70	70	70	70	70	70	70
Milk, rice (gm.)	0	0	0	0	0	0	100	100	200	200	300	300	300	300
Zwieback (gm.)	0	0	0	0	0	0	0	20- 1 piece	2 pieces	2	3	3	4	5
Raw ham (gm.)	0	0	0	0	0	0	0	0	0	50	50	50	50	50
Butter (gm.)	0	0	0	0	0	0	0	0	0	20	40	40	40	40
Calories	280	420	637	777	966	1135	1588	1721	2138	2478	2941	2941	3007	3073

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[1] From the first to the seventh day inclusive the eggs are beaten; from the seventh to the fourteenth day inclusive one-half are beaten and one-half are cooked.

continued for ten days. The eggs are beaten up entire (with a little sugar), and the cup containing them is placed in a dish filled with ice, so that they remain cold. This food at once 'binds' the super-secreted acid and therefore mitigates the pain rapidly, and causes the vomiting, often quite troublesome, to cease. The portion of milk is increased daily per 100 cc. (℥iij), and at the same time 1 additional egg is given, so that at the end of the first week the patient is receiving 800 cc. (℥xxv) of milk and from 6 to 8 eggs. Both these foods are now continued in the same amount pro die for another week. No more than *1 liter of milk a day* is allowed at any time. Besides milk and eggs, some raw chopped meat is given from the fourth to the eighth day, usually on the sixth, 35 grams (ʒix) pro die, in small divided doses (stirred up with the eggs or given alone); the day after 70 grams (ʒxviij), and later possibly more if well digested. The patient is now able to take some rice, well cooked, and a few zwieback (softened). In the third week quite a mixed diet is tolerated, the meat being given now well cooked or lightly broiled."

All heavy foods are interdicted, as well as vegetables with husks, etc., and those tending to produce flatulence. The patient is given strict orders to masticate his food thoroughly. The table on page 229 gives the daily quantities.

The bowels are not moved, both in order to avoid any peristaltic irritation and to permit the reabsorption of blood that may have passed into the intestine. One need pay absolutely no attention to constipation in the first week, even in many cases to the end of the second. After the second week the bowels are moved with small glycerin injections or warm water, and after the third week this is done daily if a movement does not occur spontaneously. After this one tries to control the bowels by means of the food and by getting the patient to go to stool regularly.

For the anemia iron is given in the form of a soft preparation of Blaud's pills:

℞.	Ferri sulphas	10.0 gm.
	Magnesia usta	1.75 gm.
	Glycerinum,	gtt. xxx (3.6 gm.).—M.

Divide in pilulæ lx; 2 pills to be taken two or three times a day.

The pills are given as early as the sixth, eighth, or tenth day of treatment, according to need, administering them first in a macerated condition.

In severe cases arsenic is also given in the form of "Asiatic pills," each containing 0.0001 gm. of arsenous acid. The dose is gradually increased, 3 for three days, 4 for four days, up to 7 for seven days, then decreasing again, 6 for six days, etc. After the tenth day and to the sixth week bismuth compositum is substituted for the subnitrate and given three times a day before meals. The patient is usually allowed up on the twenty-eighth day and is dismissed in the sixth to the tenth week. Lenhartz reports only 8 per cent. of recurrent hemorrhages after this method of treatment as compared with 20 per cent. after the older methods. No unfavorable results were produced.

Samuel Lambert, of New York, has reported favorable results.

Senator's Method.—Senator has modified Lenhartz's treatment and employs a nourishing diet, non-irritating, which tends to check hemorrhage. It consists chiefly at first of gelatin, fat, and sugar. At first a 10 per cent. sweetened gelatin solution is given in tablespoonful doses every fifteen minutes to two hours. Small amounts of fresh butter and cream are allowed, the butter given in small frozen balls and the cream beaten up with sugar to form whipped cream. The daily allowance is from 900 to 1000 calories and may be begun immediately after a hemorrhage. Gelatin is later replaced by calves'-foot jelly, etc. He occasionally adds rectal feeding, though little of late. Gradual additions are made to the diet.

Schmidt[1] believes in the Lenhartz principle of feeding, but does not increase the diet as rapidly. He gives the stomach rest a few days, like Leube, and then increases the diet more rapidly by giving gelatin, eggs, butter, cream, sugar, and rice; but chopped meat and ham with caution.

My own method depends upon whether I first treat the patient during the period of hemorrhage or later.

If the hemorrhage is taking place or has just occurred, ʒj to ij (4.0–8.0) of a 5 to 10 per cent. solution of sweetened gelatin is given cold every half hour for ten to twelve hours, even if there be vomiting. During the remaining period of the first twenty-four hours the gelatin is continued, ℥ss to j (16.0–32.0), every two to three hours while the patient is awake, and in addition the whites of 2 raw eggs are beaten up and placed in a cup on ice and given in divided doses. An ice-bag is kept on for at least two weeks if hemorrhage has just occurred.

Gelatin treatment is continued for a week. Scraped beef and ham are omitted for the first two weeks and cream substituted, to obtain the calorie values. A little sugar can be beaten up with the cream.

The day following the hemorrhage milk is begun, 200 cc. (℥vj) cold, in spoonful doses, 100 gm (10 per cent. gelatin solution) cold, in divided doses—℥ss (16.0)—every three hours, and 1 raw egg beaten up and given in divided doses. These are placed in cups which are packed in ice. The milk is increased 100 cc. daily up to 1000 cc., and no more; eggs are increased daily 1 egg up to 8 a day. Sanatogen, preferably flavored, is begun on the third day (second day after hemorrhage). With the exceptions noted, the rest of the diet is after Lenhartz.

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[1] Deutsch. med. Woch., Jan. 18, 1906.

Author's Modified Table.

	Days thereafter.												
First day, that of hemorrhage.	2	3	4	5	6	7	8	9	10	11	12	13	14
Gelatin, ʒj to ij (4.0–8.0) of a 5 to 10 per cent. solution every half hour for ten to twelve hours. Then ℥ss to j (16.0–32.0) every two to three hours to total 200 to 300 gm . . .	100	100	100	100	100	100	0	0	0	0	0	0	0
Eggs[1] (2 whites) on first day; entire thereafter	1	2	3	4	5	6	7	8	8	8	8	8	8
Sugar with eggs, none	0	0	20	20	20	30	30	30	40	40	40	50	50
Milk (cc.), none	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000
Cream (cc.), none	0	0	0	0	0	25	25	25	25	25	25	25	25
Milk, rice (gm.), none	0	0	0	0	0	0	100	100	100	100	200	200	300
Zwieback (gm.), none	0	0	0	0	0	0	20- 1 piece	20	40	40	40	60	80 4 pieces
Sanatogen (gm.), none	0	10	10	10	20	20	20	20	20	20	20	20	20
Butter (gm.), none	0	0	0	0	0	0	10	20	25	25	25	50	50
Calories . . . 200 to 400	399	730	973	1272	1467	1765	2413	2476	2748	2822	3098	3349	3519

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[1] Eggs should be raw and well beaten for ten days, and after that 4 raw and 4 soft boiled.

If no recent hemorrhage, I start the diet after the method corresponding to the third day in the table on page 232. An ice-bag is kept on for two weeks if there is hemorrhage or one within a week or ten days.

Bismuth subnitrate, 2.0 to 4.0 (ʒss–j), is given in ℥ij (60.0) water t. i. d. before feedings, commencing on the second day after hemorrhage. At times I combine magnesia usta, 1.0 (gr. 15), or sod. bicarb., 1.0 (gr. 15), with the bismuth.

Tr. belladonna, ♏x (0.66) in a teaspoonful of water can be given t. i. d. for pain. I prefer to move the bowels gently by a small soapsuds enema containing ℥ij (60.0) of olive oil on the third day after hemorrhage, and thereafter every other day. If no hemorrhage has occurred recently, then a daily movement should be secured. Milk of magnesia, ʒj to ij (4.0–8.0) in ℥iv (125.0) water given on rising, or small doses of Sprudel salts, ʒj (4.0) in a glass of hot water, are of service.

On the seventh day after hemorrhage, or immediately if no hemorrhage, iron and arsenic should be given:

℞. Blaud's iron pill.................... gr. v (0.3) (made fresh)
Sod. arsen.......................... gr. $\frac{1}{50}$ (0.0013)

Pill made soft with honey and crumbled when taking, one pill t. i. d. after eating. The arsenic can be gradually increased to gr. $\frac{1}{25}$ (0.0026) sod. arsen. t. i. d.

I give no scraped beef until the commencement of third week after hemorrhage, and then increase the diet after Leube's method. The bismuth treatment should be continued, or one can substitute the nitrate of silver treatment. It is advisable to keep this up for several months as a precaution, and then continue the diet and alkaline treatment for hyperchlorhydria for a considerable period.

At the end of three weeks the patient may be allowed to sit up for a short time daily; and at the end of four weeks to begin to go outdoors for a short time. In favorable cases it is advisable not to resume work under six weeks. Some of the chronic cases either will not remain in bed or cannot, for financial reasons.

In such event we are obliged to employ careful, but liberal feeding; the use of Carlsbad salts, iron and arsenic tonics, and either the subnitrate of bismuth treatment with an alkali in addition, or nitrate of silver. The following prescriptions are of service:

℞. Bismuth subnitrate, 2.0 to 4.0 (ʒss–j),
suspended in 4 ounces (125 cc.) of water, and given t. i. d. half an hour before meals.

This is preferable to pouring the bismuth suspension through a stomach-tube.

As olive oil protects the surface and lessens the secretion, I sometimes employ the bismuth suspended in ℥j to ij (30.0–60.0) of olive oil instead of water. Alkalis should also be used.

℞. Magnesia usta........................ 2.0 (gr. 30), or
Milk of magnesia........................ 4.0 to 8.0 (ʒi–ij)

In 3 or 4 ounces (100–125 cc.) of water alone, or combined with equal quantities of soda bicarbonate.

℞. Soda bicarb.,
Magnesia usta........................ āā 10.0 (ʒiiss)
Milk-sugar........................ 2.0 (ʒss)

Dose, 2.0 (ʒss) in water t. i. d. an hour after eating.

Nitrate of silver can be employed in place of the bismuth, to be given on an empty stomach t. i. d. half an hour before meals, as advocated by Boas and Kaufmann:

℞. Argenti nitratis........................ 0.2 (gr. 3)
Aq. destil........................ 180.0 (℥vj)

Keep in dark bottle. Tablespoonful in wineglass of water t. i. d. half an hour before eating.

Occasionally lavage of stomach with 1:5000 to 1:3000 silver nitrate has been advocated once or twice a week, if there has not been a hemorrhage for some weeks; but the internal administration is safer. It should be given for two to three weeks, then discontinued and the bismuth substituted.

The alkali can be given in addition t. i. d. an hour after meals. The meals should preferably be frequent and in smaller quantities, as in hyperchlorhydria.

Tincture of belladonna, ♏x (0.66); or extract of belladonna, gr. $\frac{1}{3}$ (0.022); or atropin, gr. $\frac{1}{100}$ (.0006), may be given t. i. d. for pain or excessive secretion. It is a valuable remedy and lessens HCl.

Rarely a hypodermic of gr. $\frac{1}{4}$ to $\frac{1}{2}$ (0.016–0.032) of codein or morphin, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.0016), may be necessary for acute pain.

In cases complicated by stenosis and ectasia lavage is necessary for the fermentation, and olive oil ℥j to ij (30.0–60.0) t. i. d. before the chief meals, to aid the passage of food. The application of Rose's belt, and the patient lying on the right side for half an hour after eating, both temporarily aid in emptying the stomach.

Hemorrhage is rare in this type, the ulcer being dormant and the chief symptoms due to stenosis. Surgery, gastro-enterostomy especially, is advocated in these cases. If tetany, the same procedure is advisable.

Vomiting.—In cases described by Lenhartz, this is relieved by neutralizing the acid, and this is the best treatment. Rarely rectal feeding may be required for a few days. Bismuth subnitrate, gr. 2 (0.13), and oxalate of cerium, gr. 1 (0.065), should be given, or 1-drop doses of Fowler's solution of arsenic, four in all, an hour apart. If the vomiting continue, a single cautious lavage is a safer procedure than the risk of recurrent hemorrhage from the strain of emesis.

Pain.—Rose's belt will at times aid in alleviating the pain due to the dragging of adhesions, by the support afforded to the viscera.

Perforation.—Temporarily, rectal feeding, a hypodermic of morphin, the ice-bag, and cautious lavage after cocainizing the pharynx, as suggested by Ewald. *Immediate recourse should be had to laparotomy.*

Surgery.—The following are the indications for operation: Perforation with commencing peritonitis; local peritonitis, with or without abscess; subphrenic abscess; perigastric adhesions; ectasia due to stenosis from ulcer or spasm from its irritation; gastric tetany with ulcer.

In *recurring acute hemorrhages* the Mayos advise opening the stomach, locating the bleeding point, suturing it firmly with catgut on the inner (mucous) side, and protecting the region by mattress sutures (musculoperitoneal). Gastrojejunostomy they have not found reliable.

In 6 cases—1 case of death from hemorrhage followed gastrojejunostomy—5 cases treated by primary operation on the bleeding point, with or without excision of the ulcer, recovered.[1]

In cases of chronic ulcer, with recurrent hemorrhages, after systematic treatment for eight months to a year by the methods I have described, and if the patient is still losing ground, I advocate resort to surgery. I am aware that some surgeons are loth to operate on such cases unless there is pyloric stenosis, and claim that in such event the gastric contents will not pass through the new opening, but through the pylorus. Cannon and Blake have demonstrated this experimentally on animals. On the other hand, when a large anastomotic opening is made, this has been shown to be of practical value, especially by the posterior no-loop method.

It has been demonstrated that the pancreatic juice and bile entering the stomach aid in neutralizing the hyperacidity, and this is of service.

Wm. Mayo states that in chronic ulcer, where there is no mechanic obstruction, the result has not been as favorable. Unfortunately, we do find cases of multiple ulcers with hemorrhages from various areas and with an open and soft pylorus, such as Munro[2] reports, in which the results of surgery are not favorable; but in any event the lessening of hyperacidity through gastro-enterostomy is of value.

In cases of pyloric obstruction (stenosis) from ulcer with dilatation of the stomach, I always advocate surgical procedure, finding that though there may be temporary improvement at times quite marked under medical treatment, in every case there is subsequent relapse.

EXULCERATIO SIMPLEX (DIEULAFOY) OR SUPERFICIAL ULCERATION OF THE STOMACH

Small ulcerations of the mucous membrane of the stomach, so minute that they appear to be little more than erosions, have been observed, from which fatal hemorrhage has taken place.

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[1] Journal Amer. Med. Association, Sept. 22, 1906.

[2] Annals of Surgery, June, 1907.

The usual symptoms of ulcer have been absent and no vomiting occurred prior to the hemorrhage.

Dieulafoy was the first to minutely describe this affection.

Anatomy.—There is a superficial round or elliptic loss of substance, involving merely the mucous membrane and the muscularis mucosæ and some blood-vessels. It does not penetrate further into the coats of the organ. The defect may be from pinhead size to a quarter of a dollar in dimension; the margins are not indurated, and it may be difficult to detect even on autopsy, being concealed in some of the folds of the mucous membrane. The stomach is healthy throughout. There may be a slightly reddened zone about the area and it may be in any location.

Etiology is unknown. It may be the beginning of an ordinary ulcer, or possibly a toxic element is responsible.

Age.—Chiefly in persons from twenty-five to thirty.

Symptoms.—The patient is in perfect health, when he suddenly vomits 0.1 (½ liter) to 1 quart (liter) of blood, accompanied by melena. The hemorrhage may prove fatal. The symptoms are those of any hemorrhage: dizziness, cold extremities, collapse, and the patient may become rapidly moribund. Occasionally the patient may recover and suffer from recurrences.

Diagnosis.—Ordinary cases of gastric ulcer suffer from gastric symptoms, while these cases do not.

From latent ulcer of the stomach it cannot be differentiated.

In chronic erosions there are no hemorrhages.

Treatment.—This is the same as in hematemesis from ulcer, morphin, gr. ¼ (0.016); ice-bag over stomach; 10 per cent. gelatin solution every half hour ʒij (8.0) by mouth; lactate of calcium, gr. x (0.66), by mouth and by rectum; ergot injections, hypodermoclysis (2 per cent. gelatin), also of saline solution, etc.

Dieulafoy recommends operative procedure, suturing the bleeding point, if medical remedies fail or if the hemorrhages are recurrent.

GASTRIC EROSIONS

These are subdivided into *acute erosions* and *chronic erosions.*

ACUTE EROSIONS (HEMORRHAGIC EROSIONS)

These are small (2 to 4 mm.) abrasions of the mucosa of the stomach, in character usually multiple, and extend partly or through the layer. There is hemorrhage (hematemesis) with this type.

They occur in the newborn; in the cachexia of children; in chronic heart and arterial diseases; cirrhosis of the liver; in acute infections with the pneumococcus (Dieulafoy); and with septic organisms; also in postoperative cases, and associated with the throbbing aorta in a considerable number of neurotic women.

Treatment is of hematemesis and also of the cause.

CHRONIC EROSIONS (EROSIONS OF THE STOMACH)

An erosion is a small superficial exfoliation of the gastric mucous membrane. Erosions of the stomach have been quite frequently found at autopsy, and the subject has been discussed by Virchow, Ewald, Gerhart, and others. The latter found nothing characteristic.

Riegel notes the frequency with which small fragments of mucous membrane are washed out of the stomach, and believes it due to the tearing from this procedure. He denies its significance as a special pathologic process, but believes that at times an examination of the fragments will demonstrate the general condition of the mucous membrane.

There is even to-day some dispute as to whether the erosions of the stomach can be described as a separate clinical entity, in view of the fact that in the majority of cases there is a chronic gastritis, and only on lavage the bits of mucous membrane are secured, some believing it to be due to manipulation with the tube.

We must credit Einhorn[1] with first describing erosions of the stomach as a clinical entity. Pariser,[2] Quintard,[3] Mintz,[4] and others have described cases.

Undoubtedly, not in every case in which bits of mucous membrane are washed out of the fasting stomach have we chronic erosions. I have noted in several cases of chronic gastritis—in which unquestionably lavage was performed in an unscientific manner by the physician in attendance—the appearance of these small fragments, and yet the patient never subsequently suffered from the salient symptoms described. Traumatism was the evident cause.

Erosions may occur in acute cases. I was recently called to attend a patient with acute gastritis, having severe and persistent vomiting streaked with blood, and though I washed the stomach with greatest care, in two washings several pieces of gastric mucous membrane were found, evidently exfoliation from the acute process and violent vomiting. The case made a rapid recovery with no further symptoms.

Unrecognized cases of chronic erosions occur, but those recognized intravitam by the clinical symptoms are, so far reported, comparatively few.

Etiology.—Chronic catarrhal gastritis is the chief factor, though Einhorn reports erosions associated with hyperchlorhydria, and I have seen them once with acid gastritis.

Symptoms.—The diagnostic symptoms described by Einhorn are:

Pain, emaciation, weakness and lassitude, and the finding in the wash-water after lavage of one or more small pieces of gastric mucous membrane. There is usually decrease in acidity and free hydrochloric acid and considerable mucus (chronic gastritis).

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[1] New York Med. Record, June 23, 1894; also Journal Am. Med. Asso., May 20, 1894.

[2] Berlin klin. Wochenschr., 1900, No. 43.

[3] Arch. f. Verdauungskrankheiten, 1901.

[4] Zeitschr. f. klin. Med., Bd. 46, 1902.

Pains.—These are not intense and occur directly after meals, irrespective of the character of the food. They last one or two hours and are never severe. There are usually intervals free from pain, though rarely the pains are constant.

Emaciation.—The patients lose weight at first; the face becomes rather thin. They have not the cachexia of cancer nor the appearance of suffering as in ulcer.

Weakness.—The patient feels weak and unable to work, and likes keeping quiet, most markedly so for a period after meals. Loss of appetite is present in some cases.

If the stomach is washed out in the fasting condition one or more small pieces of gastric mucous membrane are found in the wash-water. They appear normal under the microscope, but are infiltrated with red blood-cells. This lesion is constantly found after lavage.

Blood is rarely present, though occasionally the wash-water is slightly streaked with it.

Einhorn believes the "erosions" resulting from the peeling off of the mucous membrane are responsible for the pain and tenderness, and that it has not yet been determined whether the exfoliation recurs at the same place after healing or in new regions of the stomach.

Gastric Analysis.—The hydrochloric acid secretion is, as a rule, decreased and also the total acidity. Considerable mucus is usually found. Rarely, hyperchlorhydria is present and in one case I found acid gastritis.

In washing the fasting stomach, one to four pieces of mucous membrane (0.3 to 0.4 cm.) are found. They are blood-red and under the microscope the gastric glands and red corpuscles are visible.

Prognosis.—The disease is usually of long duration, with at times intervals of improvement.

Treatment.—The diet depends on the gastric findings. The treatment is the same as for chronic gastritis, acid gastritis, or for hyperchlorhydria, depending on the case. Hydrotherapy and out-of-door exercise are of service.

If deficient hydrochloric acid, nux, comp. tinct. cinchona, and condurango are indicated; if hyperacidity, the alkalis, such as magnesia or sodium bicarbonate.

There are three methods of local treatment:

1. *Bismuth Treatment.*—Lavage every other day with milk of magnesia to dissolve mucus, and t. i. d. gr. 15 to 30 (1.0–2.0) bismuth subnitrate before meals.

2. *Nitrate of Silver Treatment.*—This is superior in many cases. It may be administered internally, gr. $\frac{1}{4}$ (0.016) in solution t. i. d. three hours after eating; lavage with an alkali every two or three days depends on the mucus; or lavage every other day with 1 : 2000 to 1 : 1000 silver nitrate, preceding it by lavage with warm water.

Einhorn recommends intragastric galvanization on one day, with nitrate of silver spray preceded by lavage on the following day, and so alternating.

He first washes the stomach with warm water, which is all removed, and then sprays the stomach with his instrument, employing 10 cc. of a 0.1 to 0.2 per cent. solution of nitrate of silver solution, employing most of it and moving the tube about. The bottle should be opened before removal of the spray.

3. The extract of the suprarenal gland (Armour and Co.) has been recommended by Einhorn. He employs it in his powder-blower and applies it every other day to the stomach, gr. 3 (0.194) at a time, instead of the silver nitrate spray. Good reports are given of this method.

CHAPTER XIII

CANCER OF THE STOMACH (CARCINOMA VENTRICULI)—OTHER TUMORS OF THE STOMACH—APPARENT TUMORS OF THE STOMACH—FOREIGN BODIES IN THE STOMACH

CANCER OF THE STOMACH (CARCINOMA VENTRICULI)

Frequency.—In an analysis of 30,000 cases of cancer, W. H. Welch, of the Johns Hopkins Hospital, finds the stomach involved in 21.4 per cent., standing next in frequency to uterine cancer. Osler states there were 150 cases of carcinoma ventriculi in 8464 patients admitted to the hospital wards. Häberlin gives 41. per cent. from 1877 to 1886 in his statistics, while Brinton places it at about 25 per cent. Häberlin demonstrated that gastric cancer is on the increase in Switzerland, and Joseph D. Bryant shows the same in the United States. Its frequency seems to vary in different countries, Griesinger having never observed it in Egypt.

Age.—Welch finds that three-fourths of his cases occurred between the ages of forty and seventy. Osler analyzes 150 cases as follows:

Between twenty and thirty years, 6 cases; from thirty to forty, 17; forty to fifty, 38; fifty to sixty, 49; sixty to seventy, 36; seventy to eighty, 4.

Fifty-eight per cent. occurred between forty and sixty, the youngest case was twenty-two. Welch's statistics show the majority of cases between forty and sixty. The maximum liability to cancer of the stomach is, therefore, between the ages of forty and sixty. Two cases of congenital carcinoma have been reported and also a few cases under the age of thirty.

Sex.—Welch finds cancer of the stomach slightly more frequent among men, 1233 men to 981 women; and Osler in 150 cases, 126 males and 24 females. Statistics vary somewhat regarding percentages, but it seems to preponderate in males.

Race.—Among 150 cases at the Johns Hopkins Hospital, there were 131 among the whites and 19 among the blacks. The ratio of colored patients to white, however, in our hospitals is small.

Heredity.—Since several members of one family have been afflicted with cancer, many are inclined to believe heredity plays a part. Delafield[1] and Prudden hold that "while the influence of heredity is difficult to estimate, there are a few well authenticated cases of the remarkable prevalence of malignant tumors in families within a few generations."

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[1] Handbook of Pathologic Anatomy and Histology.

The statistics of Williams showed that in 235 cases of carcinoma of the uterus or breast, 9 per cent. gave a history of carcinoma in the father or mother, while in nearly 20 per cent. there was evidence of carcinoma in the family; though such statistics are suggestive and indicate that an hereditary predisposition to the development of tumor may exist, this does not account for the immediate excitement of the growth of tumors and is, as Menèstrier has urged, but one of the examples of hereditary disposition which is observed in many forms of disease, such as infections, cerebral apoplexy, etc.

Williams cites the fact that the father, brother, and two sisters of Napoleon died of cancer of the stomach, to which he himself succumbed.

William S. Bainbridge,[1] who has extensively investigated the question, believes that the hereditary acquirement of cancer requires much more study before definite conclusions can be formulated, and in this I agree.

Etiology.—Traumatism has been given as a frequent cause of cancer of the stomach, but Osler reports only 1 case in his series. Probably attention is drawn to this part by reason of the injury, and the tumor is thus recognized more early. Cider, sour wines, mental worry, and nervous strain have been suggested as predisposing causes, but they have no influence.

As the muscular fibers of the cardia and pylorus undergo frequent expansion and contraction, and are subject to more work than other portions of the stomach, Brinton believes the necessarily increased nutrition of these parts may favor glandular proliferation and be productive of a neoplasm.

Some consider chronic inflammatory disease of the mucous membrane of the stomach to be a predisposing factor in the production of carcinoma, notably the polypoid form of chronic gastritis (Menèstrier). As a rule, carcinoma develops without a previous history of long-standing gastric disturbance, and I agree with Ewald and Einhorn in believing these conditions have no influence.

The gastritis found with cancer is a secondary condition. The development of cancer on an ulcer scar has been clinically demonstrated by Hauser. Häberlin places about 7 per cent. of cases as occurring in this manner, while Moynihan gives 60 per cent., and William Mayo 36 per cent.

Regarding the parasitic origin of cancer and its infectious nature there is much dispute. Scheurlein believed he had discovered a bacillus, but later researches demonstrated his error.

Gaylord, Park, and Adami hold to the parasitic theory, and Gaylord states that in all the organs, including the blood taken from cases dying of cancer, certain organisms (parasites) are found. He has inoculated guinea-pigs and dogs with peritoneal fluid from a human abdominal tumor and produced adenocarcinoma in the lung and liver. Coley has secured, in a few cases, good results in the treatment of cases chiefly of sarcomata and of a few cases of carcinoma by the injection of blood-serum of horses treated by erysipelas cocci, and I have personally seen a favorable result in 1 case of kidney sarcoma.

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[1] Boston Medical and Surgical Journal, June 27, 1907.

Psorosperms have been found in cancer cells, but it has not yet been proved whether they are real psorosperms or dried-up and changed cells. Beard's recent theory and his suggestion of the use of trypsin in the treatment of cancer is still in the experimental stage. We must confess, so far the origin of cancer has not yet been determined, though I believe it will eventually be demonstrated to be due to some bacillus or parasite.

Morbid Anatomy.—Waldeyer demonstrated that cancer of the stomach originated from the glandular structure of the mucous membrane, being an atypic proliferation of the epithelium of the glands. Beginning in the mucosa, it infiltrates the submucosa, the muscular coat, and extends to the serosa. Early in the development of the disease the lymphatic glands become enlarged, especially those of the lesser curvature. Metastatic growths may take place. The cancerous growth, especially of a certain type, may slough and form irregular ulcers.

Varieties.—The most common varieties of cancer are the cylindric-celled adenocarcinoma (or epithelioma) and the encephaloid or medullary carcinoma; next in frequency is the scirrhus, and least frequent, the colloid cancer.

1. *Adenocarcinoma (Cylindric-celled) or Epithelioma.*—This type forms soft tumors, of firmer consistency than the medullary type, and sloughing more slowly. Microscopically, the section shows elongated tubular spaces filled with columnar epithelium, and the intervening stroma is abundant. Gradually the tubular spaces develop into cell-nests. There is frequently infiltration of the connective tissue with white blood-corpuscles. Cystic degeneration is quite common. Metastases and hemorrhage may occur.

2. *Medullary Carcinoma.*—This occurs in soft, spongy, fungating masses, which involve all the coats of the stomach and usually ulcerate early. It is large and often flat, projecting above the mucous membrane and may form villous-like projections, or a cauliflower-like outgrowth. It is soft and grayish or yellowish white and contains many blood-vessels and cells. Microscopically, it shows scanty stroma, enclosing alveoli containing irregular polyhedral and cylindric cells. It is often blackish in color, due to hemorrhage (melanotic), and has a tendency to ulcerate. Metastases are frequent.

3. *Scirrhus (Fibrous) Carcinoma.*—This is characterized by great hardness, due to abundance of stroma and the limited amount of

alveolar structure. The large amount of connective tissue makes the tumor very firm and compact. It cuts almost like cartilage, and on section has a yellow or grayish-white appearance. There is little tendency to ulcerate, except at a late stage superficially, and secondary metastases are not common. It is seen quite frequently at the pylorus, there being a diffuse thickening and hardening of the wall and then a contraction, being a common cause of stenosis. The tumor may be diffuse, involving all parts of the stomach, when it may be difficult to recognize it microscopically from cirrhosis ventriculi. It has occurred in the stomach secondary to ovarian cancer, and as a part of a diffuse carcinomatosis, with involvement of the small and large intestines. It may be combined with the medullary form.

4. *Colloid Carcinoma.*—This type of cancer is peculiar from the fact that it invades widely all the coats of the stomach. It spreads with great frequency to the neighboring parts, and at times causes secondary growths of the same nature in other organs.

The appearance on section is distinctive, showing large alveoli filled with translucent gelatinous colloid material. This is often present, even to the naked eye. On scraping, no cancer juice exudes, but gelatinous fragments.

Various transitional forms, from one variety to another, are often found. Rarely a carcinoma consisting of squamous epithelium may extend from the esophagus into the cardia.

Brinton, in analyzing 180 cases of cancer, finds the scirrhus type to be most common (72 per cent.), the medullary next in frequency; though other observers, notably Osler, consider the epithelioma to be most frequent.

Cancer of the stomach is usually primary, though secondary growths have been reported.

Cancer may also extend from the liver.

Types of Growth.—Medullary and colloid cancers involve large areas of mucous membrane, growing little above the surface, being somewhat flattened, with occasional rough nodulous masses. Blood extravasations and adhesions to neighboring organs are of frequent occurrence.

The scirrhus variety extends usually only over a small portion of the mucosa and may develop extensively in thickness, growing in depth and height. The latter type, however, occasionally infiltrates the entire stomach, causing a contraction of the organ (cancer atrophicans).

Secondary Changes in the Mucous Membrane of the Stomach.—Hammerschlag has investigated the gastric mucosa in cases of carcinoma by examining fresh pieces of mucous membrane removed in cases of resection of the pylorus at the time of gastro-enterostomy, examining also the section of the stomach.

When the hydrochloric acid secretion was intact, there were no changes in the mucous membrane. When it was absent and lactic acid present, changes occurred, and there were found destruction of the rennet glands in certain areas and small-celled infiltration and formation of connective tissue in the gastric mucosa, also mucoid changes or cystic degeneration; in effect, a secondary atrophic gastritis. Eosinophile cells are present. Hypertrophy of the muscular fibers and connective tissue has been observed. Ewald has also noted that the entire mucosa may show the lesions of chronic gastritis. It is interesting to learn that Fenwick has demonstrated that atrophic gastritis may occur in carcinoma of the breast and uterus.

Location of the Cancer.—The development of the cancer may take place in various regions of the stomach, at the pylorus or cardia or within the organ, causing variation in the symptoms according to location, and in each case necessitating a special plan of treatment.

Welch's analysis of 1300 cases is as follows: Pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; the whole or greater part of the stomach, 61; multiple tumors, 45; greater curvature, 34; anterior wall, 38; fundus, 19.

In Brinton's cases, 60 per cent. were found at the pylorus. The latter is evidently the point of selection.

Wm. Mayo believes 70 per cent. involve the pyloric region, and that 60 per cent. originate in the pylorus or within 3 inches of it.

Changes in the Shape of the Stomach.—If the cancer is situated at the cardia, the stomach is usually retracted and small in size, while the esophagus above the stricture is dilated. If the tumor constricts the pylorus, the stomach will be dilated. Gastroptosis of varying degree may be present by reason of the weight of the tumor dragging down the pylorus, and it may even lie down in the pelvis. Adhesions may distort the shape of the organ, and Riegel reports an hour-glass contraction of the stomach resulting from cancer.

Perforation.—Perforations into other viscera, through the skin, or into the general peritoneal cavity are rare. The aorta has been perforated. Subphrenic abscess has been produced. Perforation, however, seldom occurs.

Cancerous Metastases.—In an analysis of 1574 cases by Welch, metastases occurred in the lymphatic glands in 551; in the liver, 475; in the peritoneum, omentum, and intestine, 357; in the pancreas, 122; in the pleura and lung, 98; in the spleen, 26; in the brain and meninges, 6; in other parts, 92.

The abdominal lymph-glands are usually affected, but the *cervical and inguinal glands* are sometimes attacked.

Secondary growths occur sometimes at the navel or in the skin in the immediate vicinity. Infection occurs either by the blood-vessels or lymph-channels. Direct extension by continuity may take place.

The medullary and colloid types of carcinoma are often associated with metastases. Sometimes numerous small cancerous deposits occur in the pleura. The microscope will differentiate them from tuberculosis, though they have occurred together. Rarely metastases are found in the eyes.

Symptoms.—There are general symptoms and special symptoms, depending on the location of the growth.

General Symptoms.—Usually a patient of middle age, fifty to sixty years, up to a short time previous—a few months or so—being in perfect health and having had no gastric symptoms, will begin to complain of slight dyspeptic disturbance, loss of appetite, and fulness, pressure, and discomfort after eating. Belching occurs, also more or less loss of sleep and loss of strength—the symptoms looking much like a mild gastritis. The tongue is usually thickly coated. The symptoms gradually become more marked. Rarely the attack begins more acutely. The feeling of discomfort gradually merges into pain. This is generally not of the severe spasmodic type of ulcer, but is continuous in character, there not being the intermissions of freedom as in ulcer. It may remit somewhat. The pain is at times increased by the food, but is often intense at a later period after eating than in ulcer. With the belching there is at first regurgitation of food, later vomiting, usually not after every meal, but once or twice a day. This is a prominent symptom when the growth causes a stenosis of the pylorus.

I have had a patient at the Red Cross Hospital with carcinoma of the greater curvature and body of the stomach, an inoperable case who has never vomited at all, the motor function being fairly good.

Later, hematemesis occurs, generally several times in succession, and the vomitus is of coffee-ground appearance and not large in quantity. The tumor usually becomes palpable at this time, though often earlier. The patient has, meanwhile, been steadily losing weight, and this loss becomes more and more marked and anemia and cachexia are prominent. He becomes more weak and prostrated and finally dies of inanition or of complications.

These general symptoms are modified by the position of the growth. A brief analysis of the symptoms is advisable.

Anorexia, or loss of appetite, occurs in about 85 per cent. of the cases, and it seems, as a rule, to be progressive. There is at times a special aversion to meat. Riegel believes that in the early stages while the motor power remains undisturbed, the appetite remains good. This would seem as if toxemia were a factor. Boas reports fair or increased appetite in some cases, believing loss of appetite to be due to lack of care of the mouth and tongue. The toxemic theory of loss of appetite seems most logical.

Pain.—Pain is the most *constant symptom*, Osler reporting it in 130 out of 150 cases, Brinton finds it in 92 per cent. of his patients, and others report a higher percentage. It usually begins at an early date, generally in the epigastrium, but may be referred to the hypochondriac regions, the sternum, or sometimes extends to the shoulders or back. It may be lancinating or of a dull, gnawing, or burning character. There may be tenderness on pressure in the epigastrium. The pain does not occur in paroxysms and is not relieved by vomiting, as in ulcer. It is continuous and never entirely disappears, though it may remit. As a rule, it is less intense than in ulcer. It is not relieved at the end of gastric digestion. In some cases it may occur more markedly after eating, though it is not especially influenced thereby. In other cases there may be more of a painful dull feeling and it is not circumscribed as in ulcer. Sometimes over the region of the tumor the pain is most intense, as is the tenderness. Head believes there are areas of skin tenderness between the nipple and umbilicus in front and the fifth to the twelfth dorsal vertebræ behind. Exacerbations of pain are caused by ulceration of the growths or by formation of adhesions.

Vomiting.—It may come on early, but more usually later. Osler reports it in 128 cases out of 150; and Brinton in 87 per cent. At first it occurs at rather long intervals, but later may be present several times a day. It is more frequent *when the pylorus is involved*, and may come on some hours after the ingestion of food, or at times on rising, when there may be mucus or undigested food in the vomitus. It may be offensive in odor or contain changed blood, micro-organisms (Boas-Oppler bacilli), and isolated yeast-cells; but rarely sarcinæ, which are *most common in benign stenosis*. Vomiting can occur, even if the orifices are not involved; if the cardia is affected, then regurgitation is characteristic.

Extensive involvement of the anterior or posterior wall or fundus may be present without vomiting.

Hemorrhage occurred in 36 of Osler's cases out of 150; while Brinton places it at 42 per cent. Riegel believes these percentages too low, as hemorrhage may be occult, or often not examined for in the vomitus or stool. The blood is occasionally ejected in sufficient quantity to be visible. It is more frequently mixed with gastric juice, food and mucus, and presents a blackish, brownish, or coffee-ground appearance; is rarely bright red, Osler finding it in 3 cases. The quantity is much less than with ulcer, though frequent small hemorrhages may occur. Blood in the stool (melena) may accompany it, though this is rarer than in ulcer. A fatal hemorrhage seldom occurs.

Loss of Weight and Cachexia.—Progressive emaciation, especially if the disease is running a rapid course or has existed for some time, is a consistent feature. In the early stages we may occasionally see patients who appear fairly healthy, or have little loss of weight, or at times temporary improvement may occur under treatment. Unfortunately, this is evanescent, and progressive emaciation takes place. In the later stages this is marked, as are the sallow skin and peculiar ashy and cachetic appearance, with loss of strength proportionate to the loss of weight.

The Blood.—Anemia is present in a large proportion of cases; when pyloric stenosis occurs with dilatation and insufficient water absorption, on account of the concentration of the blood the number of red cells may not be greatly reduced.

The average count in 59 of Osler's cases was 3712, 186 per cmm. Average of the hemoglobin was 44.9 per cent.

Schneyer[1] has shown that normal digestive leukocytosis is absent in gastric cancer, and that the number of leukocytes during digestion and fasting is the same. Osler claims that only 54 per cent. gave positive reaction.

Leukocytosis is present in gastric carcinoma, usually of mild degree, and rarely above 12,000 to 15,000. Eosinophilia is usually present and suggestive. We find some cases without apparent tumor, in which the blood count is so low as to be suggestive of pernicious anemia, but the absence of megaloblasts and the presence of leukocytosis speak for cancer.

Tumor.—In connection with the symptoms, the presence of a tumor in the gastric region is a reliable diagnostic point. If large and superficial, it is readily detected. In Osler's 150 cases, tumor was detectable in 115. The methods of determination are as follows:

1. *Inspection.*—Position should be dorsal and the knees flexed to relax the abdomen. In some cases a protrusion can be seen in the gastric region below the ensiform or at the margin of the ribs. If there is dilatation, the lower curvature of the stomach may appear as an arched line below the umbilicus, moving up and down during respiration. Peristaltic movements are present with stenosis. With gastroptosis, the lesser curvature may be seen at times and the tumor situated thereon moving during respiration. If the tumor is at the pylorus, it may draw the stomach downward and the protrusion may be seen low down in the abdomen or even at the pelvic brim. Pulsation from the aorta may be transmitted to the tumor. Intrinsic movements in the hypertrophied muscularis may cause the tumor to appear and disappear. A subcutaneous umbilical nodule can at times be observed.

Inspection with the patient standing, as suggested by Knapp, should be carried out in any case, as the tumor can at times be thus more readily appreciated. Frequentlv, simple inspection gives no information.

2. *Percussion.*—There is dulness on percussion, or a dull tympanitic note over the tumor, which can be differentiated from the surrounding tympanites. Auscultatory percussion for its determination is described and illustrated in Chapter V, pages 75 and 76.

3. *Palpation.*—This is usually quite reliable. It determines the

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[1] Berliner klin. Wochenschr., 1894, No. 41.

position and size of the growth, whether hard and nodular, or smooth, its respiratory motility, and whether it is painful.

It is difficult to recognize a tumor on the posterior stomach wall unless it is thin and the stomach empty. On the lesser curvature, with the stomach in the normal position, it can only be felt on forced inspiration.

4. *Respiratory Motility.*—Tumors of the curvatures show greater respiratory motility than those of the pylorus. When the latter is adherent to the liver, it follows the excursion of the diaphragm.

Tumors are *smaller to the palpating finger* than they are found to be on operation.

5. *Inflation.*—This is of value in aiding inspection and also in determining whether the growth is connected with the stomach, if it be adherent, and in some cases the position of the tumor in the organ. Air can be employed for the purpose or, more easily, carbonic acid gas by the method already described.

If the tumor lies in close contact with the liver and moves away from it during inflation, the diagnosis of tumor of the stomach is evident, and the liver and gall-bladder are excluded. If this does not occur, there may be adhesions or involvement of both organs. If the tumor changes its position during inflation, there are probably no marked adhesions with the neighboring organs, an important fact in reference to operative procedure.

A tumor of the pylorus generally moves to the right and downward on inflation; and if held in this position by the hand will not ascend during expiration—expiratory fixation (Minkowski). If adherent to the liver, it will move upward.

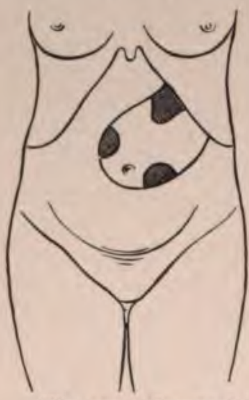

Fig. 127.—Composite from three patients showing dark areas in transilluminated stomach, produced by carcinomata of the curvatures and pylorus.

Tumors of the posterior wall and lesser curvature that are palpable before inflation are frequently no longer so thereafter. With gastroptosis, however, the tumor would be palpable, but lie higher up.

A tumor of the greater curvature descends when the stomach is inflated and occupies the lowest border of the area of inflation; it is freely movable on respiration.

The position of the tumor should be marked on the *abdominal wall before inflation* for a basis of comparison. Inflation of the colon sometimes aids in the location of the growth.

6. *Transillumination of the Stomach.*—This method is of value for the early recognition of tumors, but only if they lie on the anterior surface, on the curvatures, or at the pylorus. With the circumscribing gastrodiaphane the lesser curvature can be explored. The

method with fluorescent media is preferable. The tumor being opaque, appears as a dark spot projecting into, or within, the transilluminated area; on top, when the lesser curvature is involved; below, if the greater curvature; to the right, if the pylorus (Fig. 127).

Temperature.—This is not a regular symptom, but often appears in the later stages. It occurred in 74 of Osler's 150 cases. It is of an intermittent type and rarely runs over 101° F. Chills have occasionally been associated. Fever is probably due to some inflammatory process, or to toxic absorption from the growth.

Constipation occurs in the majority of cases and is obstinate and marked; occasionally there is diarrhea, due to food decomposition or to scybalæ, causing irritation of the intestinal canal, or as a terminal symptom due to sloughing of the cancer.

Coma similar to diabetic coma may occur, and is believed to be due to acid intoxication.

Thrombosis of the femoral vein is an occasional symptom. Osler reports general thrombosis of the superficial veins in one case.

Edema.—Swelling of the ankles frequently occurs toward the close, ascites and general anasarca sometimes are present, and the latter may appear early.

Metastases have been described. A small nodule appearing at or near the umbilicus, though rather rare, may aid in the diagnosis.

Multiple neuritis is an occasional complication.

Perforation is rare.

Tetany is a rare complication, but has occurred with malignant stenosis of the pylorus with ectasia.

Urine.—Excessive nitrogen execretion has been found in some cases, but is not constant. Diminution of the chlorids is quite frequent, while indicanuria is common. Glycosuria, acetonuria, and peptonuria have been described.

Peptonuria indicates absorption from an ulcerated area. Nephritis is often present, but would be expected in advanced age.

Special Symptoms Produced by the Location of the Growth.—(*a*) *Cancer of the Cardia.*—One of the first and an important symptom is *dysphagia*. The patient finds that there is some impediment to the entrance of solid food into the stomach and assists it by drinking water. The condition gradually grows worse. Later it is impossible to take solid food at all, as it sticks in the esophagus, causing much discomfort, and is finally regurgitated, often with considerable straining or retching. Finally, most of the fluid even is returned. There are often severe pains behind the end of the sternum and burning sensations. Mucus is sometimes ejected with the food, and occasionally blood. The cervical lymph-glands are often enlarged. Bronchitis or bronchopneumonia are frequent terminal events.

Physical Examination.—The stomach is of normal size or, in some cases, contracted; the swallowing sound is occasionally absent or heard in fifteen to twenty seconds instead of in the normal period of seven, though this is not invariably true.

Examination of the esophagus, *preferably with soft stomach-tubes of different sizes*, should be made. The position of the stricture can be noted by marking on the tube at the teeth when resistance to its passage is first encountered, and measuring the distance from the mark to the tip of the tube. *Smaller tubes* are then employed, until one of sufficiently small caliber is secured to enter the stomach.

In this way the degree of stenosis can be determined. Much force should never be used. In some cases it may be necessary to employ a stiffer tube of silkworm or bougies of varying sizes, of which Einhorn's divisible instrument is convenient (Fig. 128).

The soft stomach-tube, with openings at the tip and side, is of value for safety; by pinching the tube before withdrawal, blood, either fresh or decomposed, of foul odor or mixed with mucus, may be found within it, which in conjunction with the other symptoms is suggestive of malignancy. Occasionally a small tumor fragment may be removed, which should be submitted to microscopic examination. With malignant stricture of the esophagus, dilatation above the point of stenosis and *retention of food are associated*. It also occurs in benign stricture from syphilis or from traumatism from burns by acids or alkalis or with congenital stenosis of the esophagus, a rare condition.

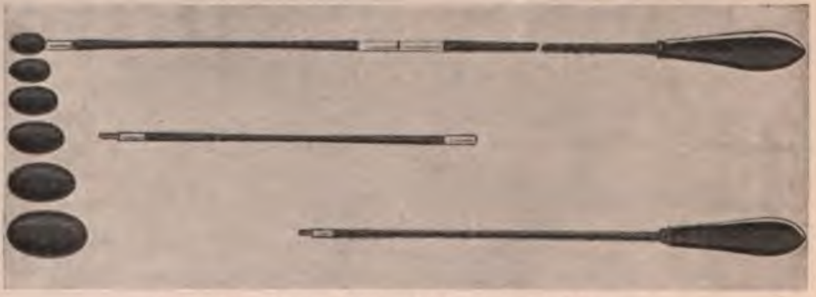

Fig. 128.—Einhorn's divisible esophageal bougie.

After the ingestion of suspended bismuth, examination with the *x*-rays and the fluoroscope will aid in determining the site of the stricture. This is of special value when it is not desirable to use the bougie.

Aspiration of the esophagus above the seat of stricture after a small test meal, and then the passage of a smaller tube and aspiration of the stomach contents, and a differential examination of the contents are not necessary for diagnosis, though they have been recommended.

The food removed from the esophagus would present the appearance as when swallowed, and there would be no hydrochloric acid; while in the stomach the particles would be finer, the reaction acid, and free hydrochloric acid might or might not be present. This is also true of the ferments. It has been demonstrated by Moore and Friedenwald[1] that in cancer of other organs than the stomach, there may be diminution or absence of free hydrochloric acid in the gastric contents, and this occurs at times in cancer of the cardia. The presence of *stricture* and *dysphagia*, the age of the patient, and the general symptoms are diagnostic.

With diverticula of the esophagus, which usually occur at the junction of the pharynx and gullet, there is generally a swelling in the neck, which can be diminished by pressure (the contents expressed), and symptoms of cancer are absent.

Spasm of the esophagus usually occurs in nervous patients, and the tube when passed is temporarily arrested. Larger tubes often pass more readily than the small ones. There are no symptoms of malignancy.

(*b*) *Cancer of the Pylorus.*—The chief subjective symptoms are pain, a full feeling in the stomach and other dyspeptic symptoms, and frequent attacks of vomiting. Just before emesis there are often severe exacerbations of pain, due to the contractions of the stomach and the effort to expel the contents through the pylorus.

The vomitus is generally large in amount (1 to 2 liters) and may consist of food taken the day before. Motor insufficiency is marked, the contents on aspiration being found to consist of more or less decomposed food, when the fasting stomach is examined. The particles of food are often quite large and obstruct the openings of the tube, and are difficult to remove by lavage.

Physical Examination.—Peristaltic unrest is a frequent symptom. Dilatation of the stomach is present, as determined by the methods of examination described. Gastroptosis may be present. Frequently a tumor can be detected lying in the epigastrium to the right of the median line or, if ptosis is present, at a lower level.

(*c*) *Cancer of the Body of the Stomach.*—Pain, anorexia, and other symptoms are manifested. Vomiting occurs in some, but in many *cases is absent*. The vomitus contains food and occasionally coffee-grounds, the food is more finely divided.

The tumor frequently lies to the left of the median line; but if on the upper curvature or posterior surface of the stomach, is not always detectable.

There is motor insufficiency of a slight or moderate degree due to infiltration of the muscular tissue by the growth, and occasionally, if the tumor be large and on the greater curvature, a slight dilatation.

Laboratory Diagnosis.—In conjunction with clinical symptoms, analysis of the gastric contents and microscopic examination aid in establishing the diagnosis of cancer of the stomach.

Gastric Contents.—Golding Bird, in 1842, first refers to the diminution or absence of hydrochloric acid in gastric cancer, but

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[1] New York Medical Journal, August 24, 1907.

Von der Velden, in 1879, first studied the question in a scientific manner.

For *accuracy* the vomitus should be examined; analysis of the gastric contents and microscopic examination, after the test breakfast, should be made and the stomach washed out. If small fragments of mucosa are found, these should be examined, as suggested by Hemmeter and Einhorn.

Vomitus.—Macroscopically, undigested meat-fibers and coarse food particles are found; the quantity is variable, depending upon the motor functions. Coffee-ground material is present in many cases and often a foul odor in advanced cases.

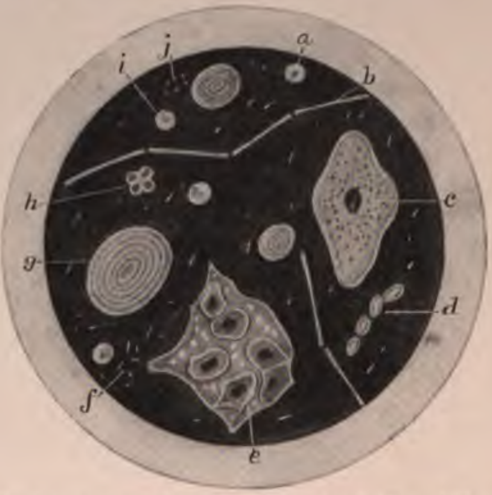

Fig. 129.—Gastric contents in carcinoma. Dark ground illumination × 460: *a*, Leukocyte; *b*, Boas-Oppler bacilli; *c*, squamous epithelium; *d*, yeast; *e*, mucous membrane fragment with carcinomatous cells as rarely found in wash-water; *f*, bacilli; g, wheat starch grain; *h*, sarcinæ (very rare); *i*, fat-droplet; *j*, cocci.

Microscopically, undigested muscle-fibers, remnants of vegetable material, starch granules, fat droplets, numerous fungi; *sarcinæ are rare;* yeast-cells are found in stagnating contents, though usually as isolated specimens; and blood and Boas-Oppler bacilli (Fig. 129). Boas and Strauss report pus[1] in some cases.

Occult blood should be tested for by Weber's or the benzidin or aloin test, if none appear microscopically.

Examination of Test Breakfast.—Ewald's or Boas' test breakfast should be given, and one hour later the contents aspirated and examined. Repeated analyses should be made.

1. *Hydrochloric Acid.*—Out of 94 of Osler's cases of gastric cancer, in 84 free hydrochloric acid was absent; and in 40 cases,

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[1] Pus may be found in benign ulceration, as in Connor's case, as well as in malignant ulcer.

Boas found it absent in 35. In the first stages of gastric carcinoma, however, free HCl is diminished, not absent. A progressive diminution in the percentage of free HCl during the course of a month or more, as determined by frequent examinations, when taken in consideration with the other symptoms, is corroborative of cancer. Einhorn reports 6 cases of gastric cancer in which free hydrochloric acid was present in normal or excessive quantity.

In gastric cancer, engrafted on gastric ulcer, free HCl (or hyperchlorhydria) has been noted in the earlier stages, often with progressive diminution accompanying the increased development of the cancer. I have seen hyperchlorhydria persist in quite an advanced stage, notably in a case at the Red Cross Hospital, which was deemed inoperable. The patient, aged sixty-eight, suffered with the symptoms of gastric ulcer for a year, falling off in weight from 240 to 200 lbs. There was then a slight gain. Later he lost steadily, and when admitted to the hospital weighed 149 lbs., was extremely weak and cachectic, with a tumor on the anterior surface of the stomach, involving the greater curvature, and with the clinical symptoms of carcinoma, except the gastric findings, which were hyperacid, with a total acidity of 90+, free hydrochloric acid 70+. The position of the growth is shown in Fig. 130. There was only slight disturbance of motor function and no vomiting. Anemia, leukocytosis, and eosinophilia were present.

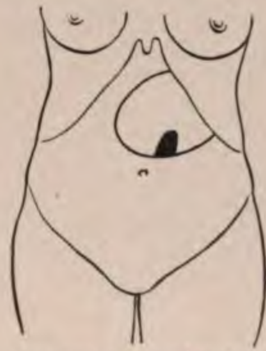

Fig. 130.—Irregular type of carcinoma under trypsin treatment at Red Cross Hospital. Carcinoma of stomach, anterior wall involving greater curvature; slight reduction of motor function. Tumor evident by palpation, percussion, and gastrodiaphany. History of ulcer with carcinoma engrafted. Hyperchlorhydria present.

The patient gained 20 lbs. in weight under trypsin treatment and proper diet, and was able to go out daily. His strength markedly improved, as well as his appearance. The growth did not disappear. The patient returned to work and at the end of several months contracted pneumonia and died.

We know that free hydrochloric acid is markedly *diminished*, or even *absent*, in severe catarrhal gastritis, and is absent in achylia gastrica. Its *absence* is not *pathognomonic to cancer*, but taken in conjunction with the clinical symptoms is confirmatory.

2. *Lactic Acid.*—It has been known for some years that organic acids were increased in cancer of the stomach and lactic acid was present; but to Boas must be given the credit of attaching diagnostic significance to it and who first described exact quantitative and qualitative methods.

He washes the stomach and gives a plate of barley soup, which contains no lactic acid, and an hour later aspirates the contents. Lactic acid should be examined for by Uffelmann's test or by Boas' method. Ewald's test breakfast will generally suffice for practical purposes.

In most cases lactic acid is present in considerable quantity, though occasionally it is absent when free hydrochloric acid is in evidence. In non-malignant stenosis with dilatation it has been found, so it cannot be said to be *absolutely pathognomonic*. The absence of free *hydrochloric acid* and the *presence of lactic acid* are confirmatory, in conjunction with other symptoms.

Boas-Oppler bacilli are found. These are rods of considerable length, frequently joined at their ends, and form long angulated threads, stainable by methylene-blue or other aniline dyes (Fig. 131). They must be distinguished from the Leptothrix buccalis (found in the mouth). A drop or two of Gram's solution should be added to the specimen. The Boas-Oppler stains brown with the iodin, the leptothrix blue.

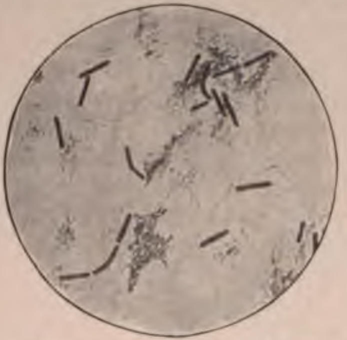

Fig. 131.—Boas-Oppler bacillus from near top of fluid from washing in case of gastric cancer. Observation at Pennsylvania Hospital (Boston).

Gram's solution consists of iodin, 1 part; potassium iodid, 2 parts; water, 300 parts.

The Boas-Oppler bacillus is found in about 80 per cent. of cases of carcinoma of the stomach; also rarely in non-malignant stenosis with dilatation of the organ.

With absence of HCl, an alkaline medium, and absence of stagnation, Paul Cohnheim also holds that the trichomonas hominis, megastoma entericum, with associated amebæ, pus, and blood, are found in carcinoma of the stomach, not affecting its motility (page 117).

Pus.—The detection of pus in the gastric contents by microscopic examination is of diagnostic value. The presence of blood, or occult blood, is also important before the tumor is palpable.

Diagnosis.—In a patient of forty-five to sixty or more years of age, suffering from recent gastric disturbances of six months' or a year's duration, or less, with a *previous history of good health;* there being continuous pain, frequently *epigastric rigidity*,[1] rapid loss of weight and strength, anorexia with or without vomiting, motor insufficiency moderate, or great with marked ectasia, with diminution or absence of free hydrochloric acid, lactic acid present; Boas-Oppler bacilli present; whether or not there be tumor detectable, or whether or not coffee-grounds are present in the vomitus, such symptoms should be considered diagnostic of carcinoma of the stomach.[1] As already noted the presence of pus and occult blood are important.

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[1] Anders, N. Y. Med. Jour., Nov. 21, 1908.

Röntgen Rays.—Anders[2] and Pfahler[3] hold that the *x*-rays aid in the early diagnosis of gastric carcinoma. The new growth makes itself evident by a change in the contour of the stomach wall; by disturbance of the peristaltic waves at certain points; at times by rigidity and contraction of the stomach wall; by adhesions which prevent the free motility of the stomach when the abdominal walls are contracted, or when the position of the patient is changed; and finally, in some cases, by obstruction to the passage of food. The method requires great care even by an expert, and may be an aid later in connection with the clinical symptoms and physical and clinical examination.

Differential Diagnosis.—*Apparent Tumors of the Stomach.*—Prolapse of the left lobe of the liver, or a pulsating aorta, or thickening of part of the abdominal muscles (recti) are referred to by Einhorn as being mistaken for a tumor or possibly for a carcinoma of the stomach. In view of the fact that with these conditions gastroptosis is usually associated, the history is a long one, emaciation is of long duration, and the symptoms of cancer are absent, the mistake can hardly occur. They are also apt to be present in younger patients. Simple adhesions of the stomach give a history most frequently of gall-bladder disease, or gastric ulcer, or localized peritonitis. Adhesions are frequently present with cancer, but the symptoms and gastric findings are of carcinoma.

Grave Anemia in Carcinoma Ventriculi, without Palpable Tumor.—These cases must be differentiated from pernicious anemia. The type occurs with mild dyspeptic symptoms. The blood-count is rarely below 2,000,000 per cubic millimeter; there is absence of megaloblasts and leukocytosis is present, which speak for cancer. There is a lower color-index, as in secondary anemia.

In addition, the acidity of the gastric contents is higher than with the achylia gastrica of pernicious anemia, and lactic acid is present in cancerous anemia. If, therefore, we have gastric symptoms, rapid loss of weight, and severe anemic symptoms in an elderly patient, the diagnosis of cancer is most probable. In secondary anemia or chlorosis, hyperchlorhydria is usually associated.

Syphilis.—This may present symptoms which may simulate carcinoma of the stomach, unless thorough examination be made. I have seen three types of this class.

1. *Sclerosis of the Stomach.*—A male patient, aged sixty, had lost 30 lbs. in weight within a period of a year; was emaciated and weak,

[1] Neusser and R. Schmidt hold that Boas-Oppler bacilli are more easily found in the stool than in the gastric contents and aid early diagnosis of cancer. Gram negative stools exclude cancer of the stomach; Gram positive stools, uniform sized Gram positive bacilli (Boas-Oppler bacilli), show cancer (Journal Am. Med. Assoc., Nov. 6, 1909, p. 1525, by P. K. Brown). I believe the Bacillus aerogenes capsulatus are found, and their deductions probably erroneous.

[2] New York Med. Jour., Nov. 21, 1908.

[3] Ibid., Aug. 21, 1909.

suffering from gastric symptoms and constipation. On examination, a small hard mass is found in the epigastrium at the left border of the ribs (Fig. 132).

It was demonstrated to be a hard and contracted stomach, giving the feel of a diffuse scirrhous carcinoma of the organ. The liver was diminished in size and hard. Free HCl were absent, the findings were of achylia gastrica. Syphilitic scars were in evidence and the patient acknowledged syphilis[1] and alcoholism.

2. *Cirrhosis of the Liver (Left Lobe—Syphilitic).*—Male, aged fifty-five, had lost 20 lbs. in a few mouths and suffered from gastric symptoms. A hard mass could be felt in the epigastrium, extending down from the lower border of the left ribs, apparently a tumor of the lesser curvature of the stomach involving the anterior wall. Respiratory movements were present. Deep palpation elicited a free edge and gastrodiaphany showed an opaque mass, but on moving the instrument a lateral view demonstrated the mass overlapping the stomach (Fig. 133).

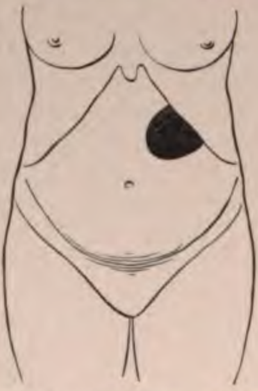

Fig. 132.—Syphilitic sclerosis of stomach. Physical examination of stomach suggests scirrhous carcinoma (diffuse) of stomach.

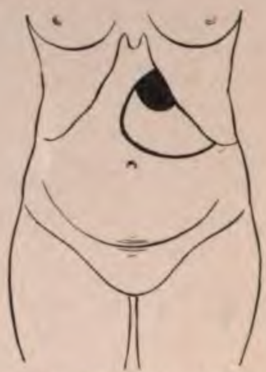

Fig. 133.—Syphilis. Enlargement of left lobe of liver overlapping stomach. Transillumination shows mass, which is readily palpable. Side view (gastrodiaphany) shows the mass not connected with the stomach. Free HCl trace; chronic gastritis.

Gastric findings were a trace of HCl and much mucus (chronic gastritis). Syphilitic scars and history were elicited. Improvement followed treatment directed to the stomach and syphilis.

3. *Syphilitic Stenosis of the Pylorus, Due to Gummatous Tumor, Simulating Malignancy.*—Male patient, aged thirty-eight. Weight, September, 1907, 196 lbs. At this time, anorexia, pain continuous in character, nausea, and occasional vomiting began. These symptoms gradually grew worse and the patient steadily lost weight. Early in November he entered a local hospital, where he was under

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[1] The demonstration of Wassermann's reaction is diagnostic in doubtful cases.

treatment by lavage, etc., for eight weeks. His weight on leaving the institution was 137 lbs. He spent two months at a western sanitarium, where he grew steadily worse, having vomited small quantities of blood on several occasions. He was given morphin for pain and subsequently contracted the habit.

At the end of March, 1908, or about six and a half months after the first appearance of symptoms, he entered the Red Cross Hospital. His weight was 119 lbs. a loss of 77 lbs., epigastric pain nearly continuous, increased some after eating, tenderness in the epigastrium, peristaltic unrest of the stomach, dilatation to one finger below umbilicus, daily vomiting. There were marked emaciation and considerable anemia, but not the cachectic appearance of cancer. No tumor was detectable by palpation or transillumination, but a sense of resistance at the pylorus. Frequent gastric analysis invariably showed free HCl absent and abundant lactic acid. Morphin was shut off, and lavage, diet, and careful observations were carried out. A syphilitic history was secured.

On account of the age of the patient, the syphilitic history and the absence of true cachexia, in spite of the gastric findings, I believed the stenosis to be non-malignant (syphilitic) and advised operation. This was duly performed. There was a hard mass on the posterior wall of the pylorus, about the size of an English walnut, blocking the passage; no glandular involvement (Fig. 134). As the patient was in poor condition, rapid gastro-enterostomy was performed. He vomited once after operation. Mercurial inunction and iodids were given, and the patient left the hospital in good condition. He would unquestionably have died from inanition unless stomach drainage had been instituted, in spite of specific treatment.

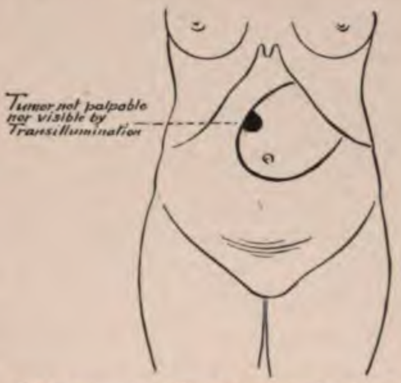

Fig. 134.—Syphilitic stenosis of pylorus due to gumma simulating malignancy. Transillumination shows dilatation, but no tumor. Growth found at operation lying on posterior wall of pylorus. Gastric findings of carcinoma, but no cachexia.

Cancer Engrafted on an Ulcer.—This condition I believe is a quite frequent occurrence. There is generally a gradual change in the symptoms and character of the gastric secretion, a gradual diminution of free HCl, though Riegel has brought to our attention that in this type the excessive production of hydrochloric acid *often persists for a long time*, and in *some rapidly fatal cases* may be present *until death*.

In these cases the diet and medication directed to the ulcer fail as soon as the malignant condition sets in. The pain increases and is continuous. There is a dislike or even loathing for food. There is rapid loss of weight and the typical cachexia appears—a change from the facies of ulcer. Hematemesis is more frequent, especially as occult hemorrhage.

This type should be considered in order to avoid error. I have already referred to a patient of this class (hyperchlorhydria), with a marked growth, cancer engrafted on an ulcer.

Aneurism of the Celiac Axis Simulating Carcinoma of the Pylorus.—The possibility of this error is interesting. A negro patient, aged forty-five, was seen by me at Roosevelt Hospital several years ago at the request of William H. Thomson.

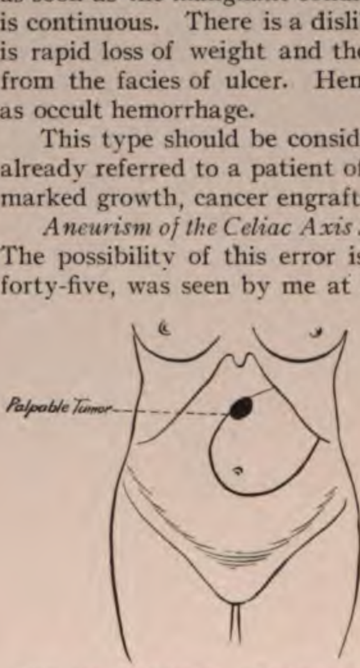

Fig. 135.—Aneurism of celiac axis with symptoms simulating carcinoma of pylorus. No tumor visible by transillumination. Tumor disappears on inflation.

The illustration (Fig. 135) shows the position of the stomach by inflation and that of the mass by palpation, it not being visible by transillumination. The tumor disappeared on inflation, diagnostic of a posterior position.

There were vomiting, gastric symptoms, such as pain, anorexia, etc., and a loss of weight of 40 lbs. in three months. The patient was very weak. Dilatation to below the umbilicus. Gastric analysis; no free hydrochloric acid; lactic acid was present.

A palpable tumor, the size of a small egg, was present in the epigastrium, in which there was slight pulsation, but no bruit or thrill. From the history and results of examination it seemed to be a carcinoma on the posterior wall, involving the pylorus, with pulsation transmitted from the aorta.

The possibility of aneurism was considered. Exploration by G. Brewer showed an aneurism of the celiac axis pressing on the pylorus posteriorly. The resulting occlusion of the pylorus and circulatory disturbances from the aneurism were responsible for the ectasia and changes in the gastric secretion.

Syphilis and aneurism must thus be considered in our differential diagnosis.

The following are the chief diagnostic points between cancer of the stomach and other conditions:

Cancer.—Age, usually forty to seventy; tongue coated; symptoms progressive and of short duration—a few months to a year; emaciation rapid; cachexia; repeated small hemorrhages, but not always; pain continuous, not paroxysmal, with no periods of relief, and not referring especially to the digestion period; anorexia; vomiting once or twice a day; later, coffee-grounds in vomitus; at times only occult blood in vomitus or stool; ectasia if the pylorus is involved; tenderness over the gastric region; no relief of pain by vomiting; anemia; free HCl diminished or absent; lactic acid present, also pus; sarcinæ rare and when present, few; Boas-Oppler bacilli in most cases; in some vomiting of blood (coffee-grounds), small in quantity and often repeated several times; melena slight. Leukocytosis moderate and eosinophilia present. Mucus present in some cases. Dysphagia and regurgitation if involvement is at cardia.

Ulcer.—Age usually twenty to forty; pain paroxysmal and worse after eating; remissions of pain, and relieved by vomiting; pain in epigastrium and in the back; local circumscribed tenderness in the epigastrium increased by pressure, at times slight tender point in the back; loss of weight; anxious expression of suffering; no cachexia; anemia; vomiting at height of digestion; appetite good; hyperchlorhydria usual; vomiting of blood in large quantity, at times of bright color; melena. Blood at times only occult.

Benign Stenosis.—Dilatation of stomach; peristaltic unrest and pain (spasmodic) preceding vomiting; long history; periods of improvement; hyperacidity usual; sarcinæ present in number; emaciation but no cachexia; vomiting of large amount; usually no tumor, and if present very small and smooth on palpation; history of previous ulcer; usually no blood, as ulcer is practically healed or contracting; anemia present; no leukocytosis.

Malignant Stenosis.—Dilatation of stomach; peristaltic unrest; short history; no period of improvement; rapid loss of weight; pain continuous; cachexia; little or no free hydrochloric acid; lactic acid; Boas-Oppler bacilli; vomiting large amount and often odorous; at times coffee-grounds in vomit from ulceration of growth; anemia marked; leukocytosis.

Chronic Gastritis.—Long history of dyspepsia; absence of cachexia; no lactic acid; less anemia; no leukocytosis; intense pain absent, more feeling of discomfort; no real pain on palpation; mucus in gastric contents, HCl diminished or absent.

Achylia Gastrica.—Total acidity much lower than in cancer, from 4+ to 2+ or less; free hydrochloric acid, pepsin and rennet absent; no mucus; scarcely any gastric juice; food particles coarse and nearly dry; course long; no cachexia; no lactic acid; may be considerable loss of weight. In transitional stage (commencing) some claim mucus is present.

Nervous Gastralgia.—Patient nervous or hysteric; pain irregular or relieved by pressure; free intervals from pain; appetite variable; no regularity of vomiting; secretory function is variable; no tumor; no cachexia; character of food makes little difference as to symptoms.

Carcinoma of the gall-bladder is found in location of gall-bladder; follows respiratory movements of the liver; shows no lateral motility and does not allow expiratory fixation. Its position is unchanged if the stomach is inflated with air. It rarely causes dilatation of the stomach unless adhesions form; and dyspeptic disturbances are not marked as a rule. Jaundice may be present. Head's zone.

Enlarged lymph-glands can hardly be mistaken, as there is the absence of gastric symptoms; no gastric findings, as in carcinoma of the stomach; and inflation of the latter organ enables a differentiation.

Growths of the peritoneum or mesentery are more diffuse and rarely movable on respiration, and gastric inflation and the symptoms enable one to differentiate.

Exudates or adhesions give none of the symptoms or gastric findings of carcinoma.

Duration of Carcinoma of the Stomach.—Osler reports 15 cases with fatality under three months; 45, under a year; 4 cases two years or over; 1 case, two and a half years. The general average is about a year to a year and a half.

Cases involving the cardia or the pylorus are more rapid, as subnitrition occurs more quickly. The medullary type is more quickly fatal. Complications shorten the disease.

It is interesting to learn, however, that some cases of inoperable carcinoma of the stomach improve greatly after palliative gastroenterostomy. Eleven such cases have been collected from Czernys'[1] clinic, in which the patients were well from two to fourteen years later.

Prognosis.—This has been considered fatal, though recent results are more favorable. Surgery has relieved conditions temporarily and prolonged life, while medical treatment has failed, though it has helped to alleviate suffering.

Kocher has reported 1 case in which the patient was in good health five and a half years after resection of the pylorus for carcinoma; and Wölfler 1 in which the patient was well for five years when a metastasis occurred.

Recently a few isolated cases of apparent cure have been reported. In an analysis of the results of operative treatment of gastric cancer at Braun's Clinic at Göttingen, Creite[2] refers to 1 case in which fourteen years after resection of the pylorus for carcinoma the patient was in perfect health.

Recently more favorable results have been reported. Leriche[3] has collected records of 89 patients on whom gastrectomy was performed, found in good health three years after operation; and of these, 34 no less than five to ten years after operation.

Out of 79 cases treated by gastrectomy, Patterson[4] collected 33 (41.6 per cent.) who were free from recurrence three years or more after operation.

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[1] Wells, "Resistance to Cancer," Jour. Amer. Med. Assoc., May 29, 1909.

[2] Journal American Medical Association, Aug. 24, 1907, p. 723.

[3] Revue de Medecin, Jan., 1907.

[4] International Medical Annual, p. 537, 1908.

Deaver shows that we may expect 10 to 15 per cent. to be cured by radical operation.

According to Kausch,[1] Makkas traced 92 of Mikulicz's patients operated on before 1902, and found 17, or 14.3 per cent., well more than three years after operation. Further statistics are unnecessary. The radical operation, gastrectomy, evidently affords results in some cases.

Schlatter reported in 1897 the first successful case of total extirpation of the stomach, with survival of the patient for a considerable period. Bernays, of St. Louis, and others have reported the survival of cases for some time after operation. The operation has been generally abandoned and we find that the so-called *gastrectomies* are generally not complete removal.

Treatment.—There are two methods of treatment, surgical and medical, of which the only hope of cure lies in the former, medical treatment being only justifiable if the case is inoperable or refuses operation or as an adjunct to palliative operation.

Surgery.—Before referring to the radical or palliative methods in surgery, I desire to call to my readers' attention the necessity of the education, not so much of the patient who will generally consent, if the matter is placed fairly before him, but of the physician and the specialist in gastric diseases, as to the value of early exploratory laparotomy for the purpose of diagnosis.

William Mayo[2] justly remarks that in an early exploratory incision we have the one diagnostic resource which is reliable and which must be resorted to in a large majority of cases before a surgical diagnosis can be made, and without it the truth is but slowly established at the expense of progressive hopeless involvement. It can be safely accomplished through a small incision. He further calls to our attention that the chemic findings of the gastric secretion gain in diagnostic importance with the progress of the disease and become of the greatest value when the patient is *in a hopeless condition* and that exploration should not be delayed by reason of the inconclusive nature of the results. He has further demonstrated that about 60 per cent. of cases of cancer begin in the pylorus and 70 per cent. in the pyloric region, and that the early diagnosis of cancer depends in a great measure upon the introduction of the *mechanic phenomena from obstruction at the pylorus.* It is the interference with gastric motility which first calls the patient's attention to his trouble and not the presence of the cancer itself. Moreover, a case with marked symptoms of cancer of the stomach, but without any evidence of pyloric obstruction, proves on exploration to be the victim of advanced and hopeless disease of the body of the organ, in which there were no symptoms during the operable period. The presence of a tumor does not demonstrate inoperability, as a small movable tumor in the pyloric region may be a favorable indication. Limitation to the pyloric end and mobility are the important factors, also the degree of lymphatic infection.

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[1] International Medical Annual, p. 537, 1908.

[2] Annals of Surgery, March, 1904.

Pyloric Stenosis is a Surgical Disease Whether it is Benign or Malignant.—It seems, therefore, especially sound doctrine in all cases with symptoms pointing to pyloric stenosis, in patients of forty years of age or over, to perform exploratory laparotomy to settle the type of stenosis and immediately further operation of greater or lesser extent, the character depending on the cause of obstruction.

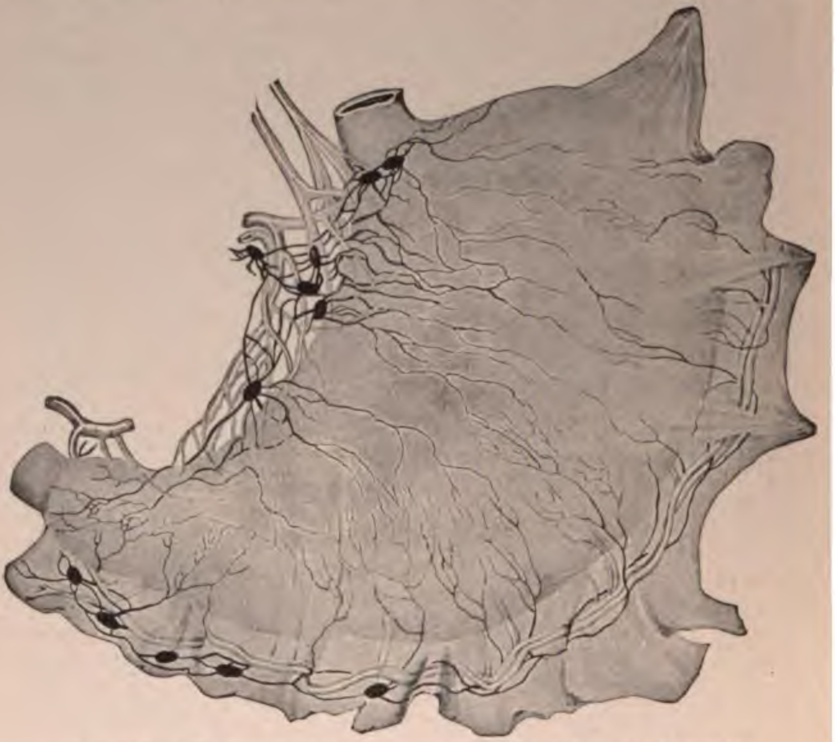

Fig. 136.—The lymphatics of the stomach (Moynihan, after Cunéo).

In elderly persons, previously in good health, with gastric symptoms and rapidly developing emaciation, after frequent examinations both of the patient and gastric contents for several weeks, even if no definite results are secured by analysis and no tumor be detectable, exploratory laparotomy is indicated. Some patients, of course, will not consent to any operation until even palliative operative procedure is too late.

I have seen a number of abdomens opened and immediately closed as inoperable, in one case notably the entire stomach wall being infiltrated. If the medical profession would recognize the value of exploratory incision, I believe many lives could be saved.

Radical Operation.—Billroth, in 1878, was the first to prove the possibility of resection of the pylorus for cancer, but it has been clearly demonstrated that this operation is insufficient. Mikulicz has pointed out that on the lesser curvature the blood and lymph-vessels lie in the wall of the stomach itself, and that it is necessary in every case of pyloric cancer to remove all the lesser curvature to the gastric artery. Cunéo has demonstrated that there are but few lymph-glands along the greater curvature and that these are confined to the pyloric region.

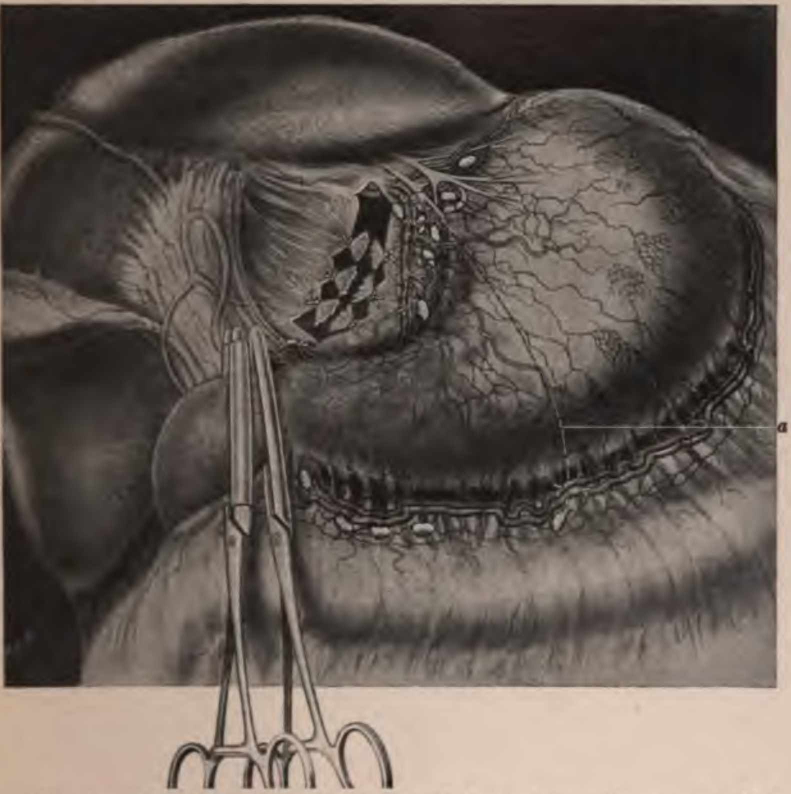

Fig. 137.—W. J. Mayo's method of partial gastrectomy for cancer of the stomach. Ligation of gastrohepatic omentum and superior vessels in such manner as to leave all the lymph-nodes attached to the part of the stomach to be excised; also lines of division of duodenum and stomach: *a*, Mikulicz-Hartmann line (Fowler).

The illustrations (Figs. 136–138) demonstrate the glandular relations, the correct line of incision, and the completed operation.

In all cases of pyloric cancer a partial gastrectomy should be performed. If cancerous metastases or marked adhesions are present, radical operation is contra-indicated; as it is in the case of extreme debility or old age.

Mayo's latest statistics show 11.45 per cent. mortality, and Deaver's 11.11 per cent. The latter has tabulated 393 cases by various operators with an average 26.5 per cent. fatality.

Palliative Operation.—This should be performed when radical operation cannot be carried out, to prevent death from starvation and to remove the irritating effect of food with the resulting pain. The operations are:

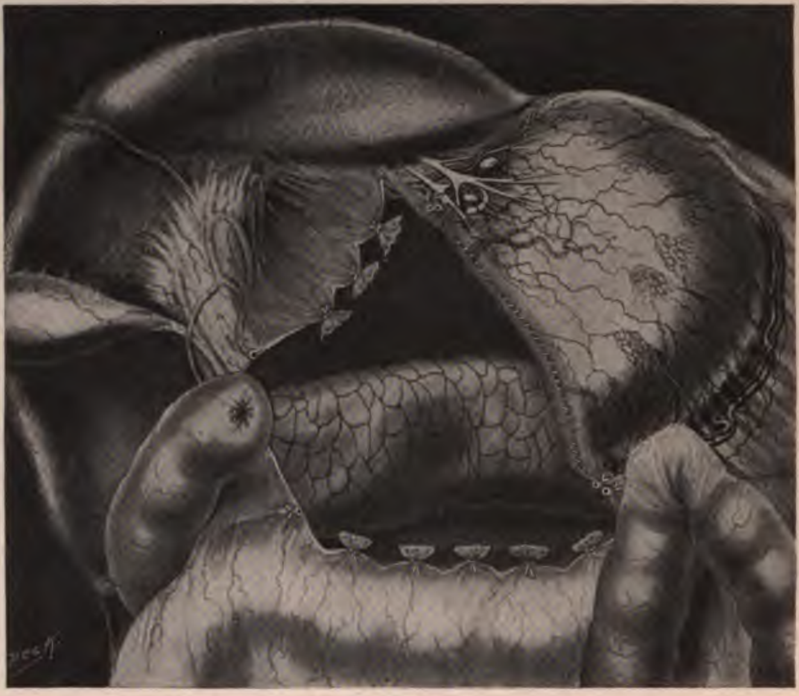

Fig. 138.—W. J. Mayo's method of partial gastrectomy for cancer of the stomach. Operation completed (Fowler).

(*a*) *Gastrostomy* is the formation of a fistulous opening into the stomach; indicated in cancerous stenosis of the cardia, with rapid loss of weight and severe pain.

(*b*) *Gastro-enterostomy* is an anastomosis between the stomach and small intestine, in cancer of the pylorus with stenosis; for similar reasons.

These operations prolong life and give the patient considerable comfort, and there is often temporary increase in weight. They are less severe than the radical operations and are only contra-indicated in extreme debility or great age, with the patient in such condition that fatality would be assured. Gastrostomy can be performed under cocain (local) anesthesia if necessary. I have already referred to the fact that in 11 of Czerny's cases the patients were living from three to fourteen years after gastro-enterostomy.

Medical Treatment.—*Diet.*—The first important feature is the endeavor to relieve the condition of subnutrition and to give the patient food which he will most easily assimilate. If there is stricture of the pylorus or esophagus, it will necessitate the use of liquid food entirely, or mushes in addition, if there is less severe obstruction.

If the cancer involve the body of the stomach alone, food of more solid character can be taken, as the motor functions are not as greatly interfered with. In the dietary we include milk, kumyss, matzoon, bacillac, farinaceous food; soups, with finely divided vegetables, such as pea, bean, and potato; broths, gruels, bouillon; raw or soft-boiled eggs; butter in plenty, tea, weak coffee, and cream; crackers softened in water and milk-toast.

In some cases, chicken, squab, scraped meat, sweetbread, stale bread, oysters, fish, etc. Sanatogen (flavored) given in ʒij (8.0) doses up to 1 ounce a day; somatose, Wyeth's beef-juice, Mosquera's beef-jelly, soluble beef peptonoids, Armour's extract of beef, tropon, and rare beef-juice are all of value.

I have employed as many as 8 raw eggs a day beaten up in milk, in addition to other foods. They possess great nutritive value. Russell's emulsion of mixed fats is of service. Food should be administered in divided small meals, four to eight a day. The calorie value can be estimated, but the chief criterion is their digestibility and the increase in the patient's weight, which should be carefully recorded. Temporary increase in weight can often be secured.

X-rays.—There have been various claims made for the value of the *x*-ray in the treatment of internal cancer, such as of the stomach or intestines, that it diminishes the size of the growth and relieves pain. I have seen cases in which the pain seemed to be somewhat relieved, but never any permanent results. In the treatment of skin cancer definite or even curative results have been secured. Beck's eventration treatment with employment of the *x*-rays is described at the end of the chapter. Incidentally Morton has recommended the use of fluorescent media internally in connection with the *x*-rays, but Henry Piffard and S. Tousey have conclusively exploded his theory and shown that fluorescence does not occur. Direct light rays are necessary.

The first researches with the internal administration and dosage of fluorescein were reported by me in connection with *gastrodiaphany*.[1]

Radium.—The radium treatment for cancer of the stomach and esophagus was first introduced by Einhorn.[2] He employs for the stomach a hard-rubber capsule that can be unscrewed and which contains a glass radium flask (Curie 20,000 strength). To the rubber capsule is attached a silk thread, in which several knots are tied, indicating the distance from the lips to the cardia and how far the capsule lies from the cardia. The capsule is introduced like his stomach-bucket. The thread is tied to the lobe of the ear and the capsule left in the stomach for one hour.

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[1] Medical News, April 10, 1904.

[2] Medical Record, March 5, 1904.

Einhorn states he has not yet formed definite conclusions as to results, but it seems to be of palliative benefit. There is no method of determining the relation of the capsule to the tumor, serious burns have resulted from prolonged exposure to radium, and no definite results are yet reported. I would, therefore, not recommend the method.

A similar instrument, except that it is connected to a thin rubber tube in which a mandrin is slipped for the purpose of introduction and then the latter removed, has been devised by Einhorn for the treatment of malignant esophageal stricture (Fig. 139).

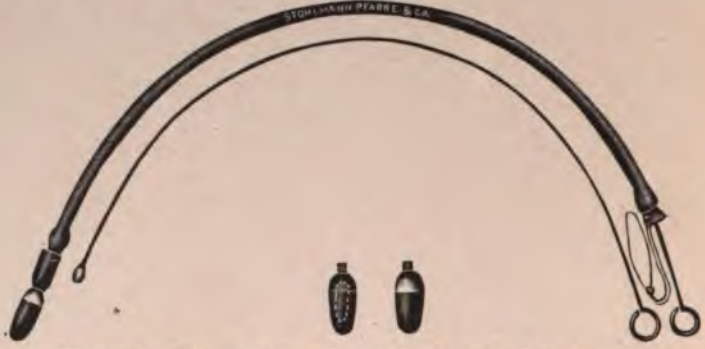

Fig. 139.—Einhorn's radium receptacle for treatment of cancerous stenosis of the cardia: Tubing, mandrin, and receptacle.

He recommends leaving it in the esophagus from half an hour to an hour, and claims an increase in the permeability of the stricture, less pain, and increased ability in swallowing. As in this case the radium can be directly applied and some definite results have been secured, the method might be of value. The question of damage from overexposure must be carefully considered. The method is evidently worthy of further investigation.

Drugs.—*Sodium Iodid.*—In stricture of the cardia, Boas has recommended sodium iodid, gm. 2.0 to 3.0 (30–45 gr.), in divided doses during the day, and claims that during a treatment of over six months the patient gained a little in weight and was relieved from some of the symptoms. Even though transient improvement occurs, it is worthy of trial if it adds to the patient's comfort. Administration for a brief period would decide the latter question.

Thiosinamin.—Sachs[1] claims to have been successful with thiosinamin by hypodermic use in the treatment of 2 cases of pyloric stricture; and Michaelis[2] found it softened an esophageal stricture and enabled him to dilate it with bougies.

Thiosinamin (fibrolysin) is moderately soluble in water, soluble in 3 parts of alcohol, and readily soluble in ether. Hypodermic injection of fibrolysin can be given in 15 per cent. alcoholic, or 10 per cent. glycerin, solution.

The average dose is gr. ½ to 1½ (0.032–0.1 gm.). Einhorn advises its use by mouth.

It is worthy of trial in stenosis of the esophagus or pylorus. The following is useful:

℞.	Thiosinamin	0.5 (7½ gr.);
	Glycerini	6.0 (ʒiss.);
	Syrup. cort. aurant	20.0 (ʒv);
	Aq. destil. q. s. ad.	60.0 (℥ij).—M.

Sig.—Teaspoonful in water t. i. d.

Arsenic has been recommended, Fowler's solution, 3 to 5 drops (0.2–0.3) daily; or sodium arsenate, gr. $\frac{1}{30}$ to $\frac{1}{25}$ (0.002–0.0026), t. i. d., but their chief value is combined with iron, such as Gude's peptomangan, or iron tropon, to combat the anemia.

℞.	Blaud's iron pill (fresh)	gr. v (0.32);
	Sod. arsen.	gr. $\frac{1}{50}$ (0.0013).—M.

One pill, made soft with honey, is an excellent combination given t. i. d.

Condurango was first recommended by Friedreich in 1874 for the treatment of cancer, but it has no specific action.

Alone or combined with dilute hydrochloric acid (suggested by Ewald), it is an excellent stomachic to improve the appetite, and at times increase of weight may result.

It may be given as the fluidextract of condurango, 15 to 20 drops (1.0–1.3), in water t. i. d., with or without dilute hydrochloric acid, half an hour before meals; or the decoction of condurango may be employed:

℞.	Decoction condurango	20.0 to 25.0 gm.;
	Water	200.0 to 250.0 cc.—M.

Tablespoonful t. i. d. before meals.

Other stomachics, such as are advised in chronic gastritis, are of service:

℞.	Acidi hydroch., dilute	ʒij (8.0);
	Tr. nuc. vomic.	ʒij (8.0);
	Comp. tinct. cinchona	℥ss (16.0);
	Aq. destil. q. s.	℥iv (125.0).—M.

Dose, one to two teaspoonfuls in water t. i. d. before meals.

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[1] Ther. d. Gegenw., 1907, No. 1.
[2] Med. Klin., 1907, No. 10.

When trypsin treatment is employed, the acids are contra-indicated.

Methylene-blue.—Einhorn[1] has employed methylene-blue in capsules once or twice daily for some years past—gr. 3 (0.2 gm.). In 1 case the tumor appeared smaller for a time and did not increase in size; for eight months the patient was free from pain and lost no further weight. Later it grew again and the case succumbed. He believes it exercises a beneficial action in some cases.

A. Jacobi[2] advocates its use in inoperable intra-abdominal cancer, having used it for about fifteen years. He reports cases of various types and claims mitigation of symptoms, prolongation of life for some years in several cases, and a temporary retrogression of the tumor. He does not claim to have absolutely cured a case. He believes that exposure to sunlight is an aid, as methylene-blue is fluorescent.

He advocates doses (divided), commencing at 2 grains a day and increasing to 6 grains, combining belladonna, and suggests the following:

℞.	Methylene-blue	gr. vj (0.4);
	Ext. belladonna	gr. $\frac{3}{4}$ (0.048);
	Arsen. acid	gr. $\frac{1}{10}$ (0.0065).—M.

Divide into four pills.

Sig.—One t. i. d. after eating and at bedtime.

Methylene-blue treatment may be tried in inoperable cases as a *palliative.*

Trypsin Treatment.—For the theory of Beard, on which he bases his so-called trypsin treatment, I refer my readers to his various articles. There have been cases of apparent cures reported, and others of complete failure.

Rice[3] reports a case of supposed cancer of the larynx with cure; while Wm. S. Bainbridge[4] demonstrated a reported cure by Morton to be *an absolute failure.*

Shaw-Mackenzie[5] and Carter[6] report cures in apparently hopeless cases, while Ball and Thomas[7] report unfavorably in 11 cases. There are many other reports for and against its use. Cleaves believes this treatment to be justified, noting a relief of pain, an improvement in metabolism, and improved mental condition. She described a tryptoglycogenic reaction as occurring from its use, consisting of a moderate leukocytosis and eosinophilia.

[1] Deutsch. med. Wochenschr., 1891, No. 18.
[2] Journal Am. Med. Association, Nov. 10, 1906.
[3] Med. Record, Nov. 24, 1906.
[4] New York Med. Journal, March 2, 1907
[5] Med. Press, May 15, 1907.
[6] Med. Press, March 18, 1907.
[7] Archiv. Middlesex Hosp., vol. ix, in Prac., August, 1907.

Wm. S. Bainbridge, of New York, has had under observation about 100 cases undergoing this method of treatment, which are reported in the Medical Record of July 17, 1909.

Space will only allow me to insert a small portion of his summary in which he deduced the following:

1. The internal medication with holadin and ox-gall aids digestion and increases elimination.
2. Aiding digestion, increasing elimination by skin, kidneys, and bowels, and decreasing local absorption are the most important features of the treatment.
3. The regimé by increasing resistance may in some cases decrease the rapidity of the malignant process.
4. Control cases given sterile water plus the regimé, did as well as those on the full enzyme treatment.
5. Trypsin injection seems in some cases to cause disintegration of (liquefy) the cancerous tissue in the center of the mass, but the periphery continues to grow.
6. Amylopsin injection seems to diminish cachexia in some cases.
7. In a small percentage of cases hemoglobin improves under trypsin injection.
8. A steady increase (6 to 12 per cent.) in the eosinophiles occurred in most cases during the trypsin injections, which lessens under amylopsin injection. There was no increase when they were given by mouth.
9. The claims for eosinophilia as a test were not substantiated.
10. Beard's enzyme treatment does not check the cancerous process.
11. It does not prevent metastases.
12. It does not cure cancer.

The author's personal observations on this method of treatment will be shortly described.

"Trypsin Treatment for Cancer."—With slight changes, I have followed Bainbridge's modification of Beard's method. I have added secretin to the treatment to stimulate the pancreas.

Trypsin (Special). A solution of double strength is now employed (Fairchild's).

For First Month.—Begin with 5 minims (0.296 cc.) deep injection daily by *hypodermic.* Increase 5 minims daily up to 35 to 45 minims daily.

For Second Month.—Trypsin one day 5 minims (0.296 cc.). Amylopsin next day, 5 minims; and so on by hypodermic. Run each up with daily 5 minims, increase up to 35 to 45 minims daily.

For Third Month.—Amylopsin daily. Commence at 5 minims (0.296 cc.) by hypodermic. Run up with daily 5 minims (0.296 cc.); increase to 35 to 45 minims daily.

If symptoms of reaction, temperature, or renal disturbance, reduce to small dose, ♏ 5 to 20 (0.296–1.184 cc.), daily.

If local irritation from injection, use ichthyol and ice-bag.

Urine should be examined every two or three days. Examination for *trypsin in the urine* can be made, as described by Bainbridge.

One holadin capsule with a secretin tablet, gr. 1 (0.06), should be given one hour t. i. d. before meals throughout treatment. Ox-gall pepules, dose, 2 each night.

Avoid all acids. Give as little salt as possible.

Give *an alkaline mineral water* one and a half hours after meals t. i. d.; can employ glass of Vichy in which is dissolved ½ teaspoonful of soda bicarbonate.

Give plenty of butter, milk, eggs (raw), nourishing food, etc.

I have frequently substituted milk of magnesia, ʒj to ʒij (4.0–8.0), for the soda bicarbonate with Vichy, as it produces less gas and aids bowel action.

I shall refer briefly to 5 cases:

Case I.—At the Roosevelt Hospital. Carcinoma of the anterior wall of the stomach (inoperable). My technic for the first week was faulty, but was followed out correctly for four weeks, during which time I could see no improvement, but no apparent retrogression. The case passed out of my hands.

Case II.—Same hospital. Carcinoma of the bladder, from which a villous process was removed and examined, confirming the diagnosis. The patient suffered from considerable bladder irritation. After ten days' trypsin injection, this condition was slightly improved, and several small fragments of the villi were passed in the urine.

Beard claims a disintegration of the tumor. Whether the passage of this material was a coincidence or not, I am not prepared to state. At the end of three weeks' treatment the patient had apparently improved slightly and insisted on leaving the hospital.

These cases were unsatisfactory, as further observation was impossible.

Case III.—This patient I have already referred to as one of cancer engrafted on an ulcer. The patient, aged sixty-eight, whose weight two years ago was 240 lbs., for a year gave the history of ulcer of the stomach, with a loss of weight of 40 lbs., and subsequent improvement under treatment with an increase of 10 lbs. The pain during this period was increased on eating, and he had free periods. No hemorrhage as far as I can learn.

A year ago, weight 210 lbs., he began to grow worse; the pain became continuous and *no free periods*. He became weak and more and more emaciated; anorexia, no vomiting.

When I first examined him, the early part of April, 1908, he was extremely weak, being hardly able to walk, markedly cachectic, and anemic. His weight had fallen from 210 to 149 lbs. There was a hard nodular mass to the left of the median line, extending from the greater curvature upward on the body of the stomach. This was readily palpable and showed clearly by transillumination. Motor insufficiency was slight.

The gastric findings have always shown a total acidity varying from 90+ to 100+ and free HCl 65+ to 70+ (hyperchlorhydria); moderate leukocytosis 12,000, and a slight increase in the eosinophiles.

I have already referred to the fact that hyperchlorhydria may be present in cancer and persist even to the time of death. The patient suffered from chronic nephritis.

During the first month, under trypsin treatment, there was slight increase of leukocytosis and eosinophilia up to 9 to 11 per cent., averaging about 9 per cent.

In the second month, with trypsin alternating with amylopsin, the eosinophilia slightly diminished to about 8 per cent.

The patient, midway in the third month, with amylopsin alone injected, eosinophilia was 7 per cent.

Holadin, alkalis, etc., continued as usual. No opiates and no alcohol or heart stimulants have been employed.

The patient averages 8 raw eggs a day, milk, gruels, a moderate amount of bread with plenty of butter, sanatogen ʒj daily, etc.

The weight increased to 160 lbs., a gain of 11 lbs., the pain, which was formerly *continuous*, occurred only occasionally at night. As nothing was given for the pain, I am forced to attribute the improvement to *the specific treatment.* The patient's strength, appearance, and mental condition were greatly improved, and he walked daily in the park. The growth did not increase in size and, in fact, slightly diminished. The forced feeding undoubtedly improved the nutrition.

Treatment for hyperchlorhydria did not relieve the continuous pain, as it was carried out for a week before the trypsin treatment was begun with no results. A second course of trypsin treatment was administered, and the patient, on leaving at the end of August, weighed 169 lbs., and was in quite good physical condition, being able to exercise and having only occasional pain.

I can substantiate, in this case at least, the statements of Cleaves as to leukocytosis, eosinophilia, the relief of pain, and the mental improvement. The tumor, apparently, diminished considerably in size, but did *not disappear.* The patient returned to work and died of pneumonia two months later.

I could not attribute the improvement in nutrition in this case to the trypsin, as, in view of the location of the growth, the motor functions were excellent.

Case IV.—This patient, aged thirty-eight, male, was a steady drinker. Two years before he had an attack apparently of gastric ulcer with several hemorrhages; then was in fair condition for some months with slight gastric symptoms. Six months previous to admission to the hospital he began to lose weight and strength, the gastric symptoms became more severe, and he began later to vomit. His weight at the commencement of his last attack was 190 lbs. When I saw him in October, 1908, he weighed 120 lbs., anemia and cachexia were marked, vomiting six to eight times daily, ectasia with the lower border of the stomach three fingers below the umbilicus, a distinct hard mass palpable in the epigastrium, blood in the vomitus and stool. The pulse was very feeble, hardly detectable, and he was only able to walk with the aid of support. He was at once admitted to the Red Cross Hospital, where the visiting surgeon and one of the physicians in consultation with me agreed that even palliative gastro-enterostomy could not be risked. Lavage was performed twice each day, and forced feeding with 8 to 10 raw eggs daily, sanatogen, ℥j (64.0), in divided doses; iron tropin, ʒj (4.0) t. i. d.; peptonized milk in divided doses, 1 quart (liter) in all, butter, broths, gruels, etc. The patient only vomited twice after lavage was begun. Rose's belt was applied to elevate the stomach, and after each feeding the patient was turned on the right side, where he remained for half an hour to aid in emptying the organ. Olive oil, ℥ij (60.0), was also given twice daily for the same purpose.

On admission October, 1908: *Gastric analysis*, free HCl absent; lactic acid, large amount; Boas-Oppler; blood.

Blood.—Slight leukocytosis, 11,000; red cells, 3,200,000; hemoglobin diminished; eosinophilia slight; nephritis, probably due to alcoholism. Trypsin injections were given for a month, then for the second month trypsin alternating with amylopsin.

Leukocytosis slightly increased; eosinophiles, 9 to 11 per cent. during trypsin injection, 7 to 8 per cent. during the combination. There was a gain of 1,000,000 red cells in the course of the two months. At the end of this period a gain in weight of 20 lbs., the patient felt quite strong, and was able to walk about without assistance. His pulse was excellent.

Operation was then performed (gastro-enterostomy) on account of adherency of tumor and of many glandular enlargements.

Subsequent to operation, holadin, ox-gall pepules, and secretin with alkalis were continued, but no further injections. Forced feeding and tonics were resumed as soon as possible. The patient remained three months in the hospital after operation, gaining in all 38 lbs. Six weeks later he reported a further gain of 2 lbs. He continued the internal medication with holadin, secretin, etc. It is now nearly a year[1] since he entered the hospital *in extremis*, and the condition is the same as when he left the institution. There was *no disappearance of the tumor*, though the growth seemed slightly smaller after the injections.

Case V.—Male, aged fifty-six, history of gastric symptoms of six months' duration, original weight 155 lbs. On admission to Red Cross Hospital weight 120 lbs. Occasional vomiting, anemia, cachexia.

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[1] This patient recently began to drink and neglect himself and has lost considerable weight, is anemic and cachectic, but no vomiting. Under resumption of forced feeding and tonics he is improving.

Gastric analysis, free HCl absent; lactic acid present; Boas-Oppler; no blood. Leukocytosis slight; eosinophiles slight increase. Ectasia to one finger below umbilicus. Tumor palpable in epigastrium. Pulse quite good. Immediate operation was advised. The growth was not large, but there was considerable glandular involvement and some nodules on the liver. Palliative gastro-enterostomy was performed. On the fourth day nutritive enemata were no longer retained and the patient was given strained broths, barley-water, white of egg, etc., by mouth; later, peptonized milk, and as rapidly as possible forced feeding was begun. Two weeks after the operation trypsin treatment was commenced.

The patient ultimately gained *40 pounds in weight.*

There was *no change in the tumor*, it apparently remaining the same size. Seven months later conditions were the same.[1]

There is no excuse for abscess formation during the trypsin treatment if proper aseptic precautions are taken. I have always applied a 5 per cent. ichthyol ointment dressing over each fresh puncture for twenty-four hours thereafter as an extra precaution and have never had an abscess. The iliolumbar region, between the twelfth rib and the crest of the ilium, is the point of selection for the injections, alternating from one side to the other.

It is not necessary to employ a special syringe and pain is avoided by using the ordinary hypodermic as follows: decant from the ampule the required amount of trypsin or amylopsin into a sterile glass. If, for example, 40 drops are to be injected, fill the syringe with 20 drops, and at the end of the injection unscrew the barrel, leaving the needle *in situ*. The syringe is then refilled, screwed on to the needle at the site of puncture, and the residue injected.

Deductions.—The mental effect on the patient is excellent; pain *was relieved* in one case; eosinophilia was markedly increased by the trypsin injections, less by the alternating injections, and some increase by the amylopsin injections. I believe this feature shows *increased body-resistance*, just as the presence of eosinophilia in typhoid is a favorable symptom. In 2 cases the growth seemed to decrease in size. There *was no disappearance of the tumor* in any case. We must remember that the palliative gastro-enterostomy relieves irritation, and in some cases the growth becomes quiescent for a long period. The *forced feeding* improves nutrition. This should be continued indefinitely, together with the holadin, secretin, ox-gall, iron, and arsenic. I consider trypsin treatment justifiable in inoperable cases and in those on whom palliative operation has been performed, as it seems to produce some benefit as an adjunct. Further investigations should be carried on.

Thymus.—F. Gwyer[2] reports the use of dried and powdered thymus of the calf in doses of ʒj to iv (4.0–16.0) three or four times a day. He claims diminution of pain, reduction of size of growth, improved digestion, and diminution or arrest of the growth. Elimination is through the secretory organs and considerable reaction occurs, so it cannot be used for over three weeks. There is a certain amount of danger from auto-intoxication and the method is still under experiment, so that I would not advise its employment.

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[1] This patient, Nov. 27, 1909, is engaged in active business.

[2] Annals of Surgery, July, 1907.

Coley's fluid (erysipelas toxin) might be tried, though the results have been more favorable in inoperable sarcoma and rarely in carcinoma. I do not advocate its use.

Bier has reported some improvement in superficial cancer by injection of heterologous blood of a pig (10 to 20 cc. at a dose); and Leyden and Bergel have experimented on animals suffering from cancer with the injection of liver extracts, but these methods are also experimental.[1]

Vomiting and Ectasia.—Systematic lavage is indicated for these conditions with normal saline solution or milk of magnesia, ℥j to ℥ij (30.0–60.0) to the quart (liter) of water; if marked fermentation, resorcin, gr. xv (1.0), can be added; or glycothymolin or listerin, ʒj (4.0) to Oj (500 cc.) of water, etc.

Hemorrhage.—This is usually not severe. Morphin, gr. ¼ (0.016), by hypodermic; the ice-bag; gelatin, 5 to 10 per cent. solution, by mouth, ʒij to ℥ss (8.0–16.0), every one to two hours for twelve hours are useful; also hypodermics of ernutin, ♏v (0.296 cc.), or ergot fluidextract, and the methods described for hemorrhage under Gastric Ulcer.

Trémolière's Solution.—Gelatin (5 per cent. solution) with calcium chlorid (2 per cent.) therein. Dose, ℥ss to ℥j (30.0–60.0) by mouth, repeated every four hours, is of value. There are not the indications to relieve hyperchlorhydria as in ulcer, so rectal feeding may be instituted for twenty-four hours or longer. The ice-bag should be kept on for twenty-four hours. Gelatin thereafter (3 per cent. solution) should be used for several days, in divided doses up to ℥xij (375 cc.) in twenty-four hours. White of egg is of value on the following day, and then fluid diet, and a gradual return to the usual feeding.

Pain.—The application of heat by the hot-water bag or hot poultices *is indicated.* Boas recommends 3 to 5 drops of chloroform on ice to be given occasionally. Chloral hydrate, 3 to 5 gr. (0.2–0.3) in water, has been recommended by Ewald, but if the patient be very weak it is a dangerous remedy. Lavage will often relieve acute attacks, especially if stenosis with dilatation are present.

Orthoform or, preferably, orthoform hydrochlorid, which is more soluble in water, can be given t. i. d., gr. v to viiss (0.3–0.5).

Tincture belladonna, ♏x (0.66) t. i. d., or extract belladonna, gr. ⅓ (0.022) t. i. d., are valuable. Heat, belladonna, lavage, and orthoform should first be tried. If they fail, it may be necessary to employ codein, preferably, gr. $\frac{1}{8}$ to $\frac{1}{2}$ (0.008–0.032) by mouth, or gr. $\frac{1}{4}$ (0.016) by hypodermic; or, as a last resort, morphin, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016). In terminal cases the opiates are indicated.

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[1] Cancer vaccine (neoformans) has been recommended for injection, but further investigation is required as to its value.

Bowels.—For *constipation*, enemata of soap and water, with or without olive oil, bowel irrigation, injection of olive oil (℥viij to Oj (250 cc.–500 cc.) at night, to be retained, rhubarb pills, the cascara preparations, aloes and belladonna compound pills, purgen or phenolax tablets, and compound licorice powder, are all of service.

For *diarrhea*, the bismuth preparations, subnitrate of bismuth, 1.0 to 2.0 (gr. xv–ʒss), several times a day; or bismuth salicylate, gr. v to x (0.32–0.64), three or four times a day; or subgallate of bismuth, the same dose; or subcarb. bismuth, 1.0 (15 gr.), three or four times a day, are of value. Chalk mixture or comp. tinct. catechu or kino, in ʒss (2.0) doses, may be combined; or at times tinct, opii, ♏x (0.892), or tinct, opii camphorat., ♏xv to xxx (0.888–1.7776 cc.), may be required in addition.

Stenosis of the Cardia.—This may require cautious dilatation, preferably with soft tubes, if possible.

In conclusion, I desire to refer to an interesting communication by Carl Beck.[1] In several cases of intra-abdominal cancer one (notably of the pylorus) incision was made over the growth, and the latter stitched to the skin, making it practically cutaneous. The *x*-rays were then applied, with apparent disappearance of the growth in several cases. In some the wounds were allowed to granulate and in others the stomach was reduced after separation of adhesions. In some of the cases the report was certainly favorable. It would seem that in cases of cancer of the stomach in which extirpation is impossible, gastro-enterostomy combined with Beck's treatment, just described, the use of trypsin, and forced feeding to improve the resisting power of the patient, might give the best results.

OTHER TUMORS OF THE STOMACH

Other tumors of the stomach (excepting carcinoma) are comparatively rare. Sarcomata, lipomatoma, fibromata, and myomata have been found, and also polypoid excrescences due to proliferation of the glands.

Sarcoma is the most common of these varieties, and may be primary or secondary.

Primary myosarcoma and fibrosarcoma are generally in the form of circumscribed nodules in the stomach wall, while lymphosarcoma is flatter and infiltrates.

The tumors vary in size and form and are situated generally on the greater curvature. Metastases are frequent.

Primary lymphosarcoma appears most frequently between twenty and thirty-five years of age, while the other types occur more frequently in older subjects.

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[1] On External Röntgen Treatment of Internal Structures (Eventration Treatment), New York Med. Journal, March 27, 1909.

Symptoms of Sarcoma of the Stomach.—These appear somewhat insidiously, in some cases gradual emaciation being first noted; the stomach symptoms are practically the same as in carcinoma: loss of appetite, sour belching, a feeling of pressure and fulness, disagreeable taste, pain, vomiting, and, finally, coffee-grounds in the vomitus. Dilatation of the stomach, if pyloric involvement and tetany have been observed in this type of case.

Gastric Analysis.—Absence of free hydrochloric acid; presence of lactic acid; Boas-Oppler bacilli are frequently present.

In effect, we may say the gastric findings and symptoms are similar to carcinoma.

The methods of physical examination are the same as in carcinoma of the stomach.

Average duration is from one to one and a half years. Schlesinger and Kundrat have shown that certain factors may be utilized for the purpose of differential diagnosis.

Metastases of the skin are more frequent with sarcoma, and excision, with examination of a cutaneous nodule, when such is present, will afford positive information. Metastases in the intestine occur more frequently with lymphosarcoma, and these do not produce stricture, but dilatation; while carcinoma causes stenosis of the gut.

The lymph-glands are more swollen in sarcoma.

The *spleen* is also swollen in sarcoma, not so in carcinoma.

The tongue follicles are at times swollen and tumefied and infiltration of the tongue may occur; there is a symmetric arrangement of the ridges, nodules, and papillæ.

Harlow Brooks believes the growth usually appears at the lesser curvature of the stomach, though it may occur in other regions. It may be multiple.

Treatment.—Early surgical operation, if possible, the indication being the same as in carcinoma.

Lavage is indicated if dilatation be present. Coley's fluid (erysipelas toxin) has proved of value, especially in some cases of sarcoma, and should be tried in inoperable cases. Severe reaction and renal disturbance may follow its use, so it should be employed with caution.

The diet should be the same as in carcinoma, and the use of stomachics.

Arsenic (Fowler's solution), beginning at ♏v (0.296) and gradually increasing to ♏xv (0.888) t. i. d., can be tried in lymphosarcoma.

Benign Tumors.—Benign tumors of the stomach are a rarity and practically impossible to differentiate in many cases. The cachexia and gastric findings of malignancy would be absent.

With benign pyloric stenosis, a tumor (thickening) is palpable in some cases, but the symptoms would be of benign stenosis, cachexia absent, and usually hyperacid gastric contents.

Occasionally a small tumor has been noted lying on the greater curvature, due to enlargement of a lymph-gland secondary to an inflamed ulcer.

APPARENT TUMORS OF THE STOMACH

Einhorn[1] has described cases of apparent tumors of the abdomen which have been mistaken for tumors of the stomach, notably, prolapse of the left lobe of the liver, exposure of the aorta (abdominal), thickening of the abdominal muscles (recti), and adhesions of the lesser curvature. Enteroptosis of a considerable degree is usually associated with the first two conditions and the history is a long one. Pulsation of the aorta may be mistaken for aneurism. Cirrhosis of left lobe of liver may simulate cancer of the stomach on palpation. The gastric analysis and other symptoms will differentiate these conditions. With gastroptosis the pancreas has been palpated and mistaken for gastric tumor. In some cases a transient tumor in the region of the stomach dependent evidently on spasm, as from ulcer, has been noted. Schnitzler[2] observed such a case.

Thickening of the recti is diagnosed as follows: The thighs and knees are well flexed, and the head and shoulders elevated so as to produce marked relaxation of the abdominal muscles. It will then be possible to slip the finger-tips beneath the edges of the relaxed and thickened rectus muscle.

FOREIGN BODIES IN THE STOMACH

These may be swallowed occasionally or purposely, or may be gradually deposited in the stomach.

Among such articles are pins, needles, scarf-pins, knives, spoons, forks, artificial teeth, glass, hooks, pens, buttons, balls of hair, bits of iron, nails, lead, wood, and even the stomach-tube. Lunatics, idiots, and young children frequently swallow foreign bodies, as do trick knife-swallowers. Cases have been reported of patients who have worked with an alcoholic solution of shellac, and who had swallowed small quantities daily, with the ultimate formation of stones in the stomach. Sarcinæ ventriculi have also accumulated in large numbers and formed a tumor.

There may be local disturbances of severe type and vomiting, or if damage be done to the mucous membrane, then hemorrhage. On the other hand, there may be no disturbances at all and the foreign body, especially if of small size and smooth, may be evacuated from the bowels. The history of the case will generally give us information. If the tumor be of sufficient size and very movable, it can be at times appreciated by palpation.

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[1] Medical Record, Nov. 24, 1900.

[2] Centralblatt f. Chir., Sept. 3, 1898.

The x-rays will give information as to its presence.

Treatment.—The use of an emetic is objectionable, as a rule, unless the foreign body is extremely small and smooth. Personally, I never employ it. Mayou has recommended the use of an electromagnet inserted in a stomach-tube, there being sufficient space at the end of the tube to draw in a small metallic object which can be located by the x-rays.

Under usual conditions, the administration of constipating food, potatoes, rice, and the soft part of bread, and keeping the bowels costive for several days, so as to form a protective mass about the foreign body, is the best method of treatment. It is an error to immediately administer a catharic, as damage is done to the intestinal canal if there be any sharp edges to the object. Complications, such as perforation or inflammatory adhesions, are liable to occur if it be of any size. Intestinal obstruction may even result. If the body is of large size or serious symptoms ensue, early gastrotomy is indicated.

CHAPTER XIV

FUNCTIONAL DISEASES OF THE STOMACH

Under functional diseases of the stomach we may classify those affections in which either the secretory or motor functions of the stomach are at fault. Anatomic lesions are present in some cases and are absent in others.

The principal symptoms are due to the disorders of secretion or motility. Among these we classify hyperacidity (hyperchlorhydria), hypersecretion (gastrosuccorrhea), atony of the stomach, dilatation of the stomach, and achylia gastrica.

Many cases of hyperacidity and of hypersecretion are pure secretory neuroses, atony may also result from nervous disorders; but there are other causes for these conditions.

When there is dilatation due to stenosis of the pylorus, as from malignant tumor, the symptoms are due to the cancer and also to the relative motor insufficiency. It is difficult, therefore, to place any special disease of the stomach under a pure classification, owing to the diverse etiology. With achylia gastrica we have the loss of secretory functions of the stomach, and it may be classified under functional diseases. It is produced either by nervous influences or by anatomic changes in the gastric mucous membrane. As in many cases organic changes are present, I judged it advisable to devote to it a separate chapter.

HYPERACIDITY (HYPERCHLORHYDRIA)

(*Synonyms.*—Hyperaciditas Hydrochlorica; Superacidity.)

The term "hyperchlorhydria" should be used to designate an increased secretion of gastric juice or, more correctly, of hydrochloric acid, during the period of digestion; that is, an overproduction of this acid. Under normal conditions the free hydrochloric acid fluctuates in the stomach within certain limits, thus:

Free HCl averages between 25+ and 50+, or about .1 to .2 per cent., and the total acidity from 40+ to 65+, or .146 to .237 per cent.

We speak of hyperchlorhydria when not only the total acidity is higher than normal, but when the excess of free HCl is above normal. It is not sufficient to merely test for the total acidity, as it may be increased by organic acids. The free HCl should be determined quantitatively.

We might place a total acidity of 70+, of which the greater part consists of free hydrochloric acid, on the dividing line. A patient with such a degree of acidity and complaining of clinical symptoms may be considered a case of hyperchlorhydria.

A total acidity of 100 to 120, with free HCl, 80+ to 90+, is quite common, and much greater degrees of hyperchlorhydria have been observed.

We must remember that individual idiosyncrasies exist as to the susceptibility to free HCl. We occasionally find patients with a total acidity of 100+, the greater part of which is free HCl, who have no subjective disturbances whatever and never suffer any discomfort.

On the other hand, I have at present a patient under treatment with a total acidity of 60+ and free HCl 40+, who has all the symptoms of hyperchlorhydria.

Frequency.—It was formerly thought that in most diseases of the stomach the gastric secretion was deficient, but it has been demonstrated that the gastric juice is increased in about one-half the cases.

One does not see so many cases of hyperchlorhydria in hospital practice, except in connection with gastric ulcer, since the symptoms rarely become so severe as to require hospital treatment. As a concomitant of chlorosis it is frequently found. The gastric analysis in these cases is often neglected. Moreover, the general practitioner rarely examines the gastric contents in cases which present the symptoms of hyperacidity. It is, therefore, difficult to secure statistics as to its frequency.

Einhorn reports a trifle more than one-half his patients to be sufferers from hyperchlorhydria. Records of my private patients show that about 50 per cent. come to me for treatment for this condition.

Etiology.—No age is exempt. It is met with in adults and quite frequently in young people. In some cases it is a *neurosis*. Nervous excitement, violent emotions, sorrow, worry, and severe mental labor may be direct causes.

Neurasthenics and melancholics often suffer from it. It is a frequent concomitant of gastroptosis. In this condition the hyperchlorhydria is not the result of the nervous condition alone, but in part due to the ptosis, with resulting circulatory changes. Its relief by Rose's belt is the proof, as reported in a series of cases at the Manhattan State Hospital.[1] It occurs with mucous colic, in which case I believe gastroptosis is a factor.

Chlorosis is a frequent cause. Irritation of the mucous membrane of the stomach may be a direct factor, as from bolting the food, large quantities of cold or hot drinks, alcoholic excess, pickles, rich condiments, and insufficient mastication.

[1] Atonia Gastrica, Rose and Kemp.

Special articles of food or drink are often productive of it, such as very strong coffee. Excessive smoking and chewing of tobacco are also factors.

Cholelithiasis and nephrolithiasis are said to be causes.

Hyperchlorhydria is the most frequent accompaniment of gastric ulcer. It is more frequent among the wealthy class, such as bankers, brokers, and professional men, though it occurs among the poor. There seems to be no special predilection for either sex.

Symptoms.—They rarely appear suddenly, but usually develop gradually. They always come on after eating and never on an empty stomach, after the time for digestion has elapsed. At first the patient complains of a disagreeable sensation or an uneasy feeling about one to three hours after a meal. It may be a feeling of pressure or fulness, or of heat, or of tingling. This increases into a feeling of distress in the epigastric region, or a burning sensation after each meal. In the mild cases there is no actual pain.

In severe cases pain may be marked, with acid belching and heart-burn, and the patients suffer severely and in some cases appear almost in a state of collapse. The burning may be felt in the esophagus or along the back and is due to the eructation of the acid contents. Violent headache often accompanies it. Some claim they can feel spasmodic movements in the stomach. This is due to contraction of the pylorus and an increased peristaltic action of the organ endeavoring to overcome the obstacle. Mild attacks of pain last for a brief period of time, while the more severe attacks persist for several hours.

Vomiting occurs occasionally during the height of the cardialgic attacks. The vomitus is very acid and burning, and after its occurrence relief, as a rule, results.

In some cases the pain appears after eating certain articles of food, and patients seem to have idiosyncrasies, such as to coffee, etc. The attacks are not always directly dependent on the degree of the digestibility of the food, and at times they can eat indigestible material without discomfort; whereas at other times digestible articles of diet will cause pain. Probably the nervous element plays a part in these cases, causing undue irritation of the nerves of secretion.

Some suffer more disturbance after a small meal, while after a large meal they have no distress. This is readily explained by the fact that the larger amount of food combines with a greater portion of the free HCl secreted.

Patients when the symptoms first appear can ease their pain by taking nourishment, especially if rich in albumin, as the whites of eggs and milk or meat. It also disappears after the administration of an alkali, such as milk of magnesia (Phillips), Vichy, or soda bicarbonate. Persons living chiefly on a starch diet suffer more intense pain than those who live largely on meat and eggs; hence, the character of the food has frequently a relation to the pain.

Appetite.—In most cases the appetite is very good, and in some is greatly increased. Others are readily satiated, but the desire for food soon comes on again.

Thirst is at times increased during the active attack, but in many cases is normal. If hyperchlorhydria is complicated by dilatation of the stomach, thirst is present.

Bowels.—Constipation is, as a rule, present, though constipation and diarrhea may alternate.

Headache.—Severe headache often occurs, or even attacks of dizziness, generally during the occurrence of gastric pain.

Nutrition.—These patients generally do not lose in weight nor present the aspect of being very sick. They are rarely particularly well nourished and are often of nervous temperament or anemic. This is not invariable, as one has at times to treat stout, well-nourished men, high livers, and inveterate smokers and drinkers, who suffer from this complaint. Rarely, under improper diet loss of weight may occur.

Nervous Symptoms.—Some patients are depressed, nervous, suffer from insomnia, and are neurasthenic. Migraine occurs in others.

Urine.—Acidity of the urine may be reduced during the course of digestion. There are no characteristic features.

Physical Examination of the Stomach.—During intervals between attacks there may be nothing in evidence.

Percussion.—At the time of the attack the stomach may be distended.

Palpation.—The greater part of the gastric region will be found to be tender on pressure or even painful in some cases. The tenderness covers a large area, generally over the greater part of the stomach, often in the region of the pylorus this tenderness is accentuated.

Splashing Sound.—This can be produced after meals or after the ingestion of water, but is not present in the fasting condition.

Examination of the Stomach Contents.—This is the decisive test. If the stomach is aspirated in the fasting condition, it is found to be empty, or only a few cubic centimeters of juice can be secured.

One hour after Ewald's test breakfast or three to four hours after the Leube-Riegel test dinner the stomach contents show an extremely acid reaction, often two to three times higher than normal (from 100+ to 150+).

It is not sufficient to determine the total acidity, but the value of the free hydrochloric acid is the important feature. Only in hyperchlorhydria is it increased, and in this condition the free HCl causes the high degree of acidity; and Töpfer's test (dimethyl-amido-azobenzol) shows that the free hydrochloric acid constitutes the chief content, and is often within 10+ to 15+ of the total acidity. Mintz's method is also excellent.

The digestive power is very good; a small disk of egg-albumen will be digested in a short time, sometimes within one-half to one hour. Three to four hours after the test dinner meat will be found to be perfectly digested, while starchy substances will be at times unchanged or little altered.

Starch or erythrodextrin will be found, Lugol's solution giving a blue or dark red reaction. If, however, a small amount of starchy food be given with a large amount of albuminous material, while the HCl is entering into combination and before free hydrochloric acid appears, the normal reduction changes in the starch may occur. With large quantities of amylaceous material the brief period before the appearance of hyperchlorhydria will not allow a complete conversion of all the starch, and this tends to remain in the stomach an abnormally long time and to produce fermentation.

Absorption from the stomach is not disturbed; potassium iodid appears, if anything, more rapidly in the saliva.

Motor Function.—This is not impaired, but is often rather increased. Two hours after the test breakfast or six to seven hours after the test dinner the organ is found to be empty or nearly so. Salol appears as salicyluric acid in the urine an hour after its ingestion when tested for with ferric chlorid solution. We must remember that hyperchlorhydria untreated, with fermentation resulting from improper diet (excessive starch), may in some cases produce atony and resulting insufficiency. Hypersecretion, which may accompany hyperchlorhydria, is another factor.

Course.—It may be rapid, coming on suddenly and lasting a short time, or chronic for months to years. It often varies in the early stages and may be intermittent in its character. The attack may last for days, weeks, or even months, and then there be a free interval for a considerable period of time. It can recur without any apparent cause, or result from some mental shock or worry, or from some dietary indiscretion. Gradually the hyperchlorhydria becomes more frequent and at last the condition becomes permanent.

In rare cases the attacks appear to come on later than usual and vomiting of an acid mass of undigested food may occur, showing the contents were retained an abnormally long time. This is undoubtedly due to spasmodic closure of the pylorus from irritation by the acid contents.

Atony and even atonic dilatation may develop from this type, and hypersecretion (gastrosuccorrhea) may result in some cases.

Prognosis.—The prognosis is, as a rule, good. Many patients can be completely cured. In some very old or severe cases there is a tendency to relapses. The disease is not dangerous to life.

Those in whom the nervous element plays a part often do not readily respond to treatment and are a source of discouragement to the physician. Relapses, in spite of the greatest care and for no apparent reason, are not infrequent.

If atony or dilatation of the stomach complicate the hyperchlorhydria, the prognosis as to cure is not as favorable.

Pathology.—As the cases are not fatal, it is hence unknown. In one case, dying of intercurrent disease, a few erosions were found. It is evidently a disorder of the secretory function.

Diagnosis.—The diagnosis of hyperchlorhydria can be made from the examination of the gastric contents:

1. An hour after Ewald's test breakfast the acidity is found increased, due to the increased quantity of free hydrochloric acid.
2. In the fasting condition the stomach is found empty or nearly so. (This excludes gastrosuccorrhea.)

Furthermore, the patient suffers from certain subjective symptoms:

1. Discomfort or, *more generally, a pain* which comes on from one to three hours after a meal. This is directly dependent upon the ingestion of food. It is not a continuous pain and there are intervals of relief. The character of the food and the quantity have a bearing. Starchy food readily produces it and a light meal more than a full meal. The latter, when rich in albumin, often causes no disturbance. The pain may be "a dead, dull pain, with a gnawing sensation," or may be of a burning character. The administration of an alkali, such as Vichy or sodium bicarbonate, relieves the pain by neutralizing the acid; or milk, raw eggs, or a meat sandwich, by binding the free acid, produces the same result.
2. In others there is more the feeling of a heat or burning in the stomach or the feeling of a sour stomach.
3. Appetite and thirst are generally normal.
4. No cachexia.
5. Marked constipation is the rule.
6. Some cases are quite nervous or even neurasthenic.

Complications.—It is often necessary to determine whether atony, dilatation of the stomach, or gastroptosis are associated with the hyperchlorhydria.

Simple Atony.—Presence of the splashing sound and 100 cc. or more of gastric contents aspirated one hour or more after Ewald's test breakfast; or the splash with the presence of food in the stomach six or seven hours after the test meal; the lower border is in normal position as determined by the splash and by percussion.

Dilatation.—Splash at the umbilicus or below shows descent of lower border. Percussion alone or with inflation, or gastrodiaphany substantiate this finding; there is no descent of the upper border. Kidneys are normal in position.

Gastroptosis.—Splash at the umbilicus and below shows descent of lower border.

Movable kidney is diagnostic *if found in addition.* There is diastasis of the recti muscles; inflation or gastrodiaphany shows descent of the upper border of the stomach.

Differential Diagnosis.—*Acid Gastritis.*—Hyperacidity is present plus abundant stomach mucus.

Hypersecretion (*Gastrosuccorrhea*).—The stomach in the fasting condition contains 75 to 100 cc. or more of very acid gastric juice. The persistent appearance of 20 cc. of gastric juice in the fasting stomach is considered pathognomonic by many. Severe attacks of pain and vomiting generally occur during the night or early morning.

Biliary Colic.—Pains are later (four to five hours after meals) or independent of meals. The pains of hyperchlorhydria are dependent on the meals. Pains of biliary colic are not relieved by alkalis and they extend over the right epigastrium and hypochondrium, and frequently to the right shoulder or right axillary region. A patient with biliary colic or even after the attack has no appetite and cannot eat.

The appetite is good in hyperchlorhydria.

The gall-bladder is painful on pressure and at times swollen. Icterus may be present. Gall-stones at times are found in the stool. Leukocytosis is present in biliary colic if inflammation is associated. Sometimes the differential diagnosis is difficult. Gastric analysis may be necessary to determine it,[1] though occasionally both conditions occur together.

Ulcer of the Stomach.—Epigastric pain is intense, appears shortly after the ingestion of food; local tenderness on pressure and pain increased thereby. Pain disappears at the end of digestion. There are the dorsal pain and vomiting in many cases soon after meals.

Hematemesis occurs or occult blood[2] is found in the gastric contents or stool. Ulcer is more frequent in women.

Nervous Gastralgia.—More frequent in women from eighteen to thirty years. Pain appears without regularity, and is in no way dependent upon the meals or character of food. Is relieved by pressure. Intervals of perfect health; nervous temperament always present. Gastric analyses often varies in the same case.

Treatment.—The treatment comprises, first, the removal of the causes of hyperchlorhydria; and second, the cure of the condition itself.

Removal of the Causes.—Interdict tobacco smoking and chewing. If a cigar or cigarette holder is employed, smoking once or twice a day I believe may be harmless, as it prevents swallowing saliva impregnated with tobacco juice, the chief source of irritation in my opinion. Alcohol in every form, including wines and beers, should be prohibited. All kinds of acids, such as acetic, tartaric, or citric, should be forbidden; and all foods prepared with them, such as with vinegar or lemon juice; and all acid or acidulated drinks.

Condiments, such as pepper, ginger, horseradish, etc., pickles, mustard, paprika, nuts, acid fruits, grapefruit, and radishes, should be prohibited.

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[1] The presence of Head's gall-bladder zone may aid diagnosis.

[2] The *discovery of occult blood* is often the *determining factor* in our diagnosis.

Avoid all extremes of heat and cold in food and drink.

Thorough mastication of the food should be enjoined.

Nervous conditions, when present, should be treated.

Hygienic regulations are important.

Overwork and mental anxiety are factors in the production of hyperchlorhydria. Brokers, professional, and business men must be relieved temporarily from overwork and worry by being sent into the country, where various open-air amusements, such as golf, horseback riding, driving, walking, etc., can be indulged in. If the patient have a taste for fishing or shooting, such diversions are excellent. I have seen a few weeks in camp work wonders.

Those indulging in a continuous round of social festivities should be compelled to lead a quiet life.

On the other hand, there are many people of wealth with no occupation whatever who become entirely self-centered and nervous therefrom, and for such a class of persons occupation is of great value.

Hydrotherapy, such as sponge-baths, douches, etc., are of service; also a moderate indulgence in calisthenics (ten minutes morning and night).

For the cure of the hyperchlorhydria there are practically two methods used, alone or in combination:

We may bind the excess of free hydrochloric acid by the administration of large quantities of proteids or we may neutralize the excessive acid by the administration of an alkali. Clinical observation has demonstrated that those articles of food which are capable of binding large quantities of HCl are borne the best. The burning feeling of distress or pain is *relieved by the administration of albuminous food.* Carbohydrates, if given in any quantity, cause distress. The diet is, therefore, of greatest importance.

Diet in Hyperchlorhydria.—As noted under prophylaxis, all articles which are liable to overstimulate the secretory glands of the stomach should be forbidden. Among such are acids, all spices, as pepper and mustard, pickles, horseradish, olives, acid fruits, beers, and wines.

The food should be rich in albumin, such as chops, steak, roast beef, mutton, game, eggs, milk, oysters, and fish. Bread and butter can be taken, the former in moderation. Green vegetables, such as spinach, asparagus, lettuce, peas and string beans, potatoes, rice, and other cereals should be given in small quantity. They should be taken in combination with large amounts of albuminous food.

Alcohol in all forms, including beers and wines, I believe should be avoided, also coffee; though some allow a small amount of beer and very weak coffee. Fleiner has demonstrated that egg-albumen binds more free hydrochloric acid than any other food. Among other articles especially suitable for this purpose he recommends boiled veal, beef, mutton, raw ham, Leube-Rosenthal's beef solution, boiled ham, boiled pork, Swiss cheese, Roquefort, pumpernickel, milk, and cocoa.

In my own experience I have found gelatin[1] an excellent remedy, employing 1 or 2, or even 3 ounces of a 5 to 10 per cent. gelatin solution flavored with a pinch of sugar or a little vanilla, and given midway between meals. The value of egg-albumen and cocoa is marked. Starchy foods that have been well dextrinized, such as Zwieback and toast, are more readily digested.

Strauss has shown that if carbohydrates are introduced in the form of sugars in solution they do not markedly increase HCl secretion. He gives 200 to 300 cc. of a 20 per cent. dextrose solution during the day.

Considerable water should be taken during meals, or Apollinaris and seltzer, if no atony is present.

Fats, such as butter and cream, are of value. Since the carbohydrates are restricted and additional calories must be secured, Strauss, Ewald, and others advise their use. Furthermore, fats seem to lessen the acidity or possibly the irritable tendency of the mucous membrane. Cream has been thus recommended.

For some years I have been accustomed to administer olive oil once or even three times daily before meals in obstinate cases of hyperchlorhydria, using from ℥ss to j (16.0–32.0), suspended in water. This has lessened the hyperacidity. In the same way, glycerin, ʒss to ij (2.0–8.0), mixed in water, may be used. Illoway[2] employed it in one case.

If we attempt to treat hyperchlorhydria by diet alone, we should give three additional feedings at a time after the regular three meals, such as would bind the excessive hydrochloric acid and prevent the symptoms. The extra feedings may consist of kumyss, matzoon and Vichy, bacillac, bouillon, a sandwich, milk, raw egg (especially the whites) and milk, with crackers or bread and butter. One can select a diet from the tables with a sufficient number of calories.

For practical purposes an improvement in nutrition, *i. e.*, *some increase in weight*, should be secured, even though slight, in addition to the amelioration of symptoms. This refers only to those of thin habit, not to the well nourished. The use of the scales, weighing at stated intervals, is of radical importance. Assimilation differs in individual cases, and though on paper the calories may be correct, a *loss of weight* shows a radical error in the treatment. The following is a sample dietary, such as is usually recommended for a patient of good physique and quite active. The content of proteid is very high as compared with Chittenden's scale, which I advocate in health. It is frequently advisable to diminish the quantity of meat and substitute milk, matzoon, kumyss, or bacillac. Lactone-buttermilk is also excellent. Gelatin solutions bind the free hydrochloric acid in a satisfactory manner. Depending on the normal weight, height, and occupation, the diet must be formulated in every case:

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[1] Calves' foot or chicken jelly are excellent.

[2] New York Med. Jour., May 25, June 1, 15, and 29, 1901.

		Calories.
7.30 A. M.—	250 cc. milk, cocoa, 2 eggs, 3 zwieback, and butter (gm. 20)	690
10.30 A. M.—	200 cc. milk, with 1 raw egg, or matzoon and Vichy 125 cc., or kumyss 250 cc., or milk 250 cc., or bouillon with 1 raw egg (approximately).	240
	Bread 2 slices, or crackers 2 oz. (gm. 60)	160
	Butter gm. 20, water, or Apollinaris, or Vichy 250 cc., occasionally weak tea	163
1.30 P. M.—	Chops, steak, beef, or mutton, 100 gm. (about)	200
	Mashed potatoes, 30 gm	37
	Spinach, 30 gm	12
	Bread (1 slice), 30 gm	81
	Butter, 10 gm	80
4.30 P. M.—	Same as at 10.30 A. M.	563
7.00 P. M.—	Soup (barley), 200 cc.	100
	Meat, broiled, 100 gm	200
	Spinach or peas, 50 gm	30
	Potatoes mashed with milk, 50 gm	80
	Weak tea, 100 cc. (three-fourths milk)	64
	Toast (1 slice)	75
	Butter, 10 gm	81
10.00 P. M.—	Milk and Vichy, āā 100 cc.	64
	Total calories	2920

Various modifications may be made.

Coffee I interdict. All fried food should be forbidden, as should hot breads, hot biscuits, and rich desserts. The food should not be excessively hot or cold. The patient should eat slowly, masticate thoroughly, and rest after eating for at least twenty minutes to one-half hour.

Illoway and Bouveret advocate only three meals a day, with a sandwich at bedtime in some cases. They believe the stomach should have an interval of rest so as to become perfectly emptied. Practically, Illoway's only medication is one-half glass of French Vichy, given at 10 to 11 A. M., at 4 to 5 P. M., and at night, if the sandwich is omitted.

They believe that unless the stomach have some rest, atony or atonic dilatation may result.

On the other hand, some patients can only eat a small amount of food at a time, have the desire to eat at frequent intervals, and feel better if they do so. Repeated feedings are difficult to follow out in some cases, so conditions vary.

My method is to give the three regular meals a day with diminished carbohydrates, with the alkalis an hour to an hour and a half or even two hours later. If it is necessary to improve nutrition, additional feedings are given, a glass of kumyss, or milk and raw egg, or matzoon and Vichy or 1 to 2 ounces of 10 per cent. gelatin solution (sweetened) between meals or if pain comes on after the administration of the alkali. These foods can be alternated.

Medication.—Among the alkalis that are of value are Vichy, imported or siphon, bicarbonate of soda, milk of magnesia (Phillips), magnesia usta, magnesia ammoniophosphorica, and biborate of soda (Jaworski).

A little sugar of milk can be added to the soda or magnesia preparations to make them more agreeable. If the hyperchlorhydria is complicated by atony, then bicarbonate of soda (which generates considerable carbonic acid gas) would be objectionable.

The following rule should be carried out as regards the administration of the alkalis: "Give them in ample time to anticipate the appearance of symptoms by a considerable margin." They should be administered t. i. d. after meals, depending upon the time of appearance of symptoms. Repeat the dose if the symptoms begin later, unless albuminous food is given as a substitute.

The magnesia preparations have the tendency to move the bowels and hence are of special value. If the result is too active, combine the magnesia with varying proportions of soda bicarbonate or give the latter alone, or give bismuth subnitrate with the magnesia. Magnesia usta will neutralize about four times more acid than soda bicarbonate.

The dosages are as follows, given from one to two hours after meals t. i. d.:

Vichy	175 to 250 cc. (½–1 glassful).
Vichy	½ to 1 glassful plus sod. bicarb. ½ to 1 teaspoonful (2.0–4.0). Allow this to become flat (effervescence to disappear).
Sod. bicarb.	½ to 1 (2.0–4.0) teaspoonful in one-third glassful of water.

℞.	Magnesia usta,	
	Sod. bicarb.	āā

Dose, ½ to 1 teaspoonful (2.0–4.0) in water t. i. d.
A little sugar of milk or ♏j oil of peppermint may be added to flavor.
If the bowels move too freely, diminish magnesia, then give:

℞.	Magnesia usta	℥ss (16.0)
	Sod. bicarb.	q. s. ℥ij (64.0).—M.

Dose, ½ to 1 teaspoonful (2.0–4.0) in water.
If necessary, bismuth subnitrate, ʒij to ℥ss (8.0–16.0), may be added.

℞.	Sod. bicarb.	℥j (32.0)
	Magnesia usta	℥ss (16.0)
	Magnesia ammon. phos.	℥ss (16.0).—M.

Dose, ½ to 1 (2.0–4.0) teaspoonful in water.

℞.	Magnesia usta	℥ss (16.0)
	Pulv. rhu.	gm. 15 (1.0).
	Soda bicarb.	℥ss (16.0)
	Sugar of milk	℥ss (16.0).—M.

Dose, ½ teaspoonful (2.0) in water if costive or,

Milk of magnesia (Phillips) .... ʒj to iv (4.0–16.0), in water t. i. d.

This I have found to be an excellent preparation, especially where constipation is marked. It is one of the most serviceable remedies for hyperchlorhydria.

Sod. biborate	gr. x to xv (0.6–1.0)

in water (Jaworski).

has been recommended.

The use of Carlsbad water or a glass of the imported Carlsbad salts, ʒj to ij (4.0–8.0), in warm water on rising, lessens acidity and helps the bowels.

Wolff's artificial Carlsbad mixture consists of:

℞. Sod. sulph.	30.0
Sulph. potass.	5.0
Sod. chlorid	30.0
Sod. carb.	75.0
Sod. biborate	10.0.—M.

He adds sod. biborate on account of Jaworski's recommendation.
Dose, ʒss (2.0) in one-half glassful of warm water two hours before meals.

In some cases I give olive oil, ℥ss to j (16.0–30.0) or more, or glycerin, ʒss to ij (2.0–8.0), in a little water before meals, to lessen gastric irritability; bismuth subnitrate, gr. 15 (1.0), given with the olive oil is often useful.

If the pain is severe the administration of belladonna before secretion commences, in order to lessen the gastric juice, is of value. At the time of pain it is too late. It is serviceable also in obstinate cases, thus before meals:

℞. Tinct. belladonna	ʒiss (6.0)
Aq. destil.	q. s. ℥ij (60.0).—M.

Dose, ½ to 1 teaspoonful (2.0–4.0), or larger dose of tinct. belladonna, up to 10 drops, may be given.

or

Ext. belladonna	gr. ⅛ to ¼ (.01–.02).

or

Atropin	gr. $\frac{1}{100}$ (0.0006).

Illoway suggests:

℞. Tinct. aconite root	6 drops
Tinct. belladonna	25 drops
Aq. destil.	q. s. ℥j (30.0).—M.

Dose, 1 teaspoonful (4.0) on rising and a second dose in half an hour. No more.

If the pain is extremely severe and not relieved by an alkali, a small dose of codein, gr. ⅛ to ¼ (0.008–0.016), may be required. This may even be given by hypodermic with excellent results. Morphin in same dosage is rarely necessary and should only be given by the physician.

The application of heat to the epigastrium by moist compresses, hot-water bag, or poultice is of service.

If the patient is very restless and disturbed one of the bromids, strontium bromid, sodium bromid, or ammonium bromid, may be given in gr. x to xv (.06–1.0) doses for a brief period.

In obstinate cases, silver nitrate, gr. ⅛ to ¼ (0.008–0.016) t. i. d. or in solution, has been recommended to relieve gastric secretion, or occasionally a douche or spray with silver nitrate (1 : 2000), followed by lavage with water. If the douche is used, lavage is unnecessary unless there is a complicating atonic dilatation. If one sees the patient during a severe attack of pain, aromatic spirits of ammonia, ʒss to j (2.0–4.0), diluted well with water; lime-water, ℥j to ij (30.0–60.0), mist. cretæ, ℥ss to j (16.0–30.0), or bicarbonate of soda or the magnesia preparations can be given at once.

Sometimes sodium bicarbonate distends the stomach and causes more pain. Emesis is often a relief, and at times, when other treatment fails, lavage may be used as a temporary method.

Electricity has been recommended, especially by Einhorn, for the treatment of hyperacidity.

Internal galvanization has been suggested for the relief of pain. It seems to be impractical.

In cases when constipation is obstinate or when atony is present it might be of service, either the intragastric or, preferably, the percutaneous method of faradization.

Bowels.—Massage, vibratory massage, and rectal injections of olive oil are of service, as well as out-of-door exercise, horseback riding, etc. Remedies such as cascara, aloes, phenolax, etc., may be at times temporarily necessary.

If there is atony, dilatation, or ulcer, these conditions must be treated. Rose's belt is indicated for atony or atonic ectasia.

To briefly recapitulate. I have found the following of value:

Carlsbad salts on rising.

Milk of magnesia (Phillips) or magnesia usta, alone or with soda bicarb., or Vichy and soda bicarb., one to two hours after meals; midway between the chief meals a little milk and raw eggs, kumyss, matzoon and Vichy, or 1 to 2 ounces (30.0–60.0) of gelatin solution (5 to 10 per cent.).

In some cases olive oil before luncheon and dinner, and belladonna extract or tincture when required.

The proper diet must be followed.

GASTROSUCCORRHEA (CONTINUOUS SECRETION OF GASTRIC JUICE)

(*Synonyms.*—Hypersecretion; Parasecretion; Excessive Flow of Gastric Juice; Reichmann's Disease; Gastroxynsis—Rossbach.)

Gastrosuccorrhea is a perversion of function in which the glands of the stomach secrete large quantities of gastric juice even when the *stomach is empty*, and hence when there is no irritation from ingested food. The diagnosis rests on the removal from the stomach in the fasting condition of a considerable quantity of gastric juice, with the addition of symptoms which present a characteristic picture. To Reichmann the credit is due of having first called attention to this perversion of function.

Gastrosuccorrhea may be classified in two types:

1. Gastrosuccorrhea continua periodica or the intermittent form of hypersecretion; the attacks occur at irregular intervals.

2. Gastrosuccorrhea continua chronica or chronic hypersecretion.

Though some believe that hypersecretion is a purely secretory neurosis, other factors can also produce it. Unquestionably, nervous conditions, such as mental excitement or mental overexertion, may be the direct cause in some cases; hence, gastrosuccorrhea may be a pure neurosis.

On the other hand, direct irritation of the mucous membrane can be the cause; in fact, the same factors that produce hyperchlorhydria, such as rapid eating, indigestible food, spices and condiments, abuse of alcohol, bolting the food, excessively hot or cold food or drink, etc.

Hyperchlorhydria of long standing, especially if neglected, may be a factor in the ultimate production of continuous secretion. In some of the gastric crises of locomotor ataxia gastrosuccorrhea is at times observed.

Hypersecretion is also at times an accompaniment of dilatation of the stomach, either of the atonic type or, more frequently, in the form due to stenosis of the pylorus. It also may occur with ulcer of the stomach. It can even accompany acute dilatation of the stomach, especially in that form engrafted on a chronic stenotic dilatation, to which I refer under Acute Ectasy. Tetany may be rarely associated.

In many cases of hypersecretion disturbances of the motor functions of the stomach are present in addition.

GASTROSUCCORRHEA (CONTINUA PERIODICA)

(*Synonyms.*—Intermittent Secretion of Gastric Juice; Intermittent Hypersecretion; Gastroxynsis; Periodic Continuous Flow of Gastric Juice.)

This type of hypersecretion is characterized by an acute attack of continuous secretion of gastric juice associated with severe pains in the stomach, usually spasmodic in character, and by vomiting of acid fluid, the attacks usually occurring in the night or early morning and at irregular intervals; hence it is known as intermittent or periodic. Probably this condition is more frequent among nervous cases than we generally suppose. I have been able to absolutely determine in several cases of supposed intermittent attacks of acute gastritis, or so-called acute bilious attacks, that this condition of intermittent hypersecretion was present.

History.—Reichmann[1] was the first to describe this perversion of function, and Rossbach,[2] under the nomenclature gastroxynsis, described what is generally considered the same disease. Sahli, Riegel, and many others have written on it.

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[1] Berl. klin. Wochenschr., 1882, No. 40.

[2] Deutsch. Arch. f. klin. Med., 1883, Bd. 35.

Etiology.—In some cases it is a neurosis, or the result of mental overexcitement or of overexertion; irritation of the gastric mucous membrane, as from cold water, or smoking, spices, etc., may precipitate an attack.

It is associated with the gastric crises of locomotor ataxia in some patients, or with organic affections of the peripheral or central nervous system, with gastric ulcer, or with the stenotic form of ectasia or with acute dilatation of the stomach. These are not pure cases, but have the additional symptoms incident to the disease.

Symptoms.—These usually begin during the night. The patient, who is generally apparently perfectly well, suddenly begins to feel discomfort in the gastric region, which is rapidly followed by pain of severe type and generally spasmodic in character. There is nausea, a feeling of faintness, and the patient is obliged to assume the recumbent position. He grows pale, the extremities become cold, and the abdomen at times is sunken and the pulse rapid and feeble. The nausea becomes worse and worse and soon a violent attack of vomiting of a large amount of acid fluid takes place.

There is temporary relief, but the symptoms begin again, and after a short period the patient again vomits up a large quantity of fluid, far out of proportion to the amount previously ingested.

During the attack the appetite is lost and there is extreme thirst. Severe headache and constipation generally accompany these attacks.

These attacks generally occur in the middle of the night or early in the morning, and awaken the patient by the pain, if he be sleeping.

During the attack the stomach is tender on pressure and there is a good deal of heart-burn and acid belching, the urine is scanty, alkaline, and of high specific gravity.

Character of the Vomitus.—The fluid is watery and very acid. It may be clear or somewhat tinged with bile (yellowish green).

There may be particles of food in the first vomit if motor insufficiency is associated with the condition, but in many cases there is simply the clear gastric juice alone or mixed with bile. If this fluid be examined it will be found to contain free hydrochloric acid in considerable quantity, rennet, and pepsin. The desire to vomit frequently persists and generally several attacks of vomiting succeed each other. Even though the patient abstain from all fluid, in a few hours or less he will again vomit a large quantity of gastric juice. Rarely the vomitus may contain traces of blood, which does not necessarily mean an ulcer. This condition may last for several hours or for several days, when gradually the vomiting stops, the nausea and pain subside, and the patient begins to desire food. Gradually the appetite returns, the food is retained, in a few days he begins to feel nearly well, and in a brief time is apparently in perfect health.

The patient may continue in good health for weeks, months, or a year, and then have a recurrence. The intermissions of good health, on the other hand, may become shorter until, finally, the intermittent gastrosuccorrhea becomes chronic.

In many cases during the intermissions, if the gastric contents are analyzed, they will be found to be perfectly normal. On the other hand, some cases may suffer from mild symptoms of hyperchlorhydria, and such a condition will be found to exist on examination.

Diagnosis.—This can be made by the characteristic symptoms beginning during the night, the vomiting of pure gastric juice occurring when no ingesta are present in the stomach. Analysis shows that it possesses all the properties of the gastric juice with an excess of free hydrochloric acid. If no food is given and the stomach be aspirated before the second attack of vomiting occurs, at the time of appearance of pain and nausea, or if the second vomitus be analyzed and in either event be found to consist of a considerable quantity of pure gastric juice, the diagnosis is conclusive.

The attacks are intermittent.

All cases should be examined as to motor functions and dilatation, since attacks of gastrosuccorrhea occur with these conditions. One should also exclude organic disease, such as the gastric crisis of locomotor ataxia. In the latter case we have loss of patellar reflexes, the Argyll-Robertson pupil, and the Rhomberg symptom.

Prognosis.—In pure cases of gastrosuccorrhea continua periodica the prognosis may be fairly good. It is often possible to lessen the severity of the attacks or in some cases even to effect a cure.

Treatment.—*Prophylaxis.*—We must first endeavor to find the cause of these attacks, and by correcting it prevent their recurrence. In the interval between attacks the stomach contents should be examined after a test breakfast or meal, and we should determine whether or not hyperchlorhydria exists.

The motor functions of the stomach should also be carefully tested, and any motor insufficiency if present should receive treatment. If there be excesses in smoking or drinking, tobacco and alcohol should be cut off. If there are errors in diet, they should be corrected. If mental overexertion or nervous excitement is a cause, such conditions should be corrected. If the patient is neurasthenic, he should receive careful treatment. Hygienic method of living, exercise out of doors, horseback riding, golf, etc., are serviceable.

Treatment of the Attack.—During the early stage, when the pain and nausea first begin, binding the free acid with the whites of several raw eggs beaten up in water or in milk, or the use of ℥j to ij (30.0–60.0) of 10 per cent. gelatin, or neutralizing the acid by the administration of an alkali, such as ʒss to j (2.0–4.0) sodium bicarbonate in water,

℥iv (125.0), or Vichy; or milk of magnesia, ℥ss (16.0), or magnesia usta, ʒss to j (2.0–4.0), will at times mitigate the symptoms. Einhorn recommends a large dose of bromid at the appearance of the first symptom and claims that it will often cut the attack short or lessen its severity. About gr. 15 to 30 (1.0–2.0) of sodium bromid should be given, preferably in Vichy (an alkali).

The patient should be kept recumbent, with the application of moist heat or dry heat (wet hot flannel compress or hot-water bag) applied to the gastric region. He will generally vomit in spite of treatment.

The best method, I believe, is to perform lavage *early*, not waiting until vomiting occurs, if the *pain and nausea are not* relieved by administration of the albuminous food or alkalis.

Wash the stomach thoroughly with an alkaline solution—1 to 2 quarts (liters) of warm water in which ℥ij to iij (30.0–45.0) of milk of magnesia (Phillips) have been dissolved—or ℥ss (16.0) of sod. bicarb. or ℥ss (16.0) of magnesia usta. Before withdrawing the tube after lavage, pour through it into the stomach ʒij (8.0) of milk of magnesia dissolved in ℥ij (30.0) of water, to which is added ♏x (0.6) tincture of belladonna to check further secretion. Sod. bicarb., ʒj (4.0), may be substituted for the magnesia.

If the pain is very severe it may be necessary to give one or several hypodermics of morphin, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), preferably combined with atropin, gr. $\frac{1}{200}$ to $\frac{1}{100}$ (0.00032–0.00065), which last is of value to lessen hypersecretion. Codein may be employed.

To the use of cocain for nausea and vomiting I am opposed. It gives but temporary relief, is a marked cardiac depressant, many patients have an idiosyncrasy to even a small amount, and there is danger of the cocain habit.

It may be necessary to repeat lavage several times. The belladonna or atropin should be pushed to physiologic limits, with dilatation of the pupils and dryness of the throat, if one expects to have any effect in checking the hypersecretion.

Some recommend washing the stomach with 1 : 2000 silver nitrate solution, and following it with plain warm water.

Some patients will not consent to lavage, and in these cases we can simply give internally several large doses of the alkalis with belladonna every two to three hours, whether they vomit or not.

If there is *great thirst*, a glass of hot water in which sodium bicarbonate, ʒj (4.0), has been dissolved is of service, if taken in small amounts (teaspoonful doses). It often relieves the nausea and the alkali is of value; or a small piece of cracked ice or an occasional teaspoonful of cool water can be also employed.

The raw white of eggs beaten up, placed in a cup and packed around with ice, or the 5 to 10 per cent. gelatin, two teaspoonfuls given every half-hour or so, both relieves thirst and binds the acid; or

Milk	℥viij (250 cc.)		Pack thi in ice.
Oxalate of cerium	gr. x (0.6)	dissolved therein.	
Sodium bicarbonate	gr. 15 to ʒss (1.0–2.0)		

Give 1 teaspoonful (4.0) every half-hour or so as to relieve thirst and bind the acid, and check nausea and vomiting.

In some cases rectal enemata of hot normal salt solution at 115° F., Oj (500 cc.), relieve thirst and stimulate the pulse. It may be necessary to employ smaller quantities of saline solution or to use proctoclysis.

It is best for the patient not to drink too much the first day of the attack, except the remedies noted, as an excessive amount of fluid favors vomiting. No food should be given except as above advised. On the next day small quantities of milk diluted with lime-water or matzoon and Vichy or albumen-water can be given, ℥ss to j (16.0–32.0), every hour, and the gelatin can be kept up.

The quantity of nourishment may then gradually be increased and, finally, soft-boiled eggs, scraped raw meat, and a subsequent diet such as is used in hyperchlorhydria.

The bowels should be moved early in the attack by a soapsuds enema, 1 quart (liter), to which ℥viij (250 cc.) olive oil have been added, or by enteroclysis.

To recapitulate. In the treatment of the acute attack I follow out the following method: Heat to the abdomen, the administration at once of an alkali by mouth; the bowels are moved by enema, and the stomach, after a brief period of rest, washed with an alkali, preferably with milk of magnesia; ʒij (8.0) of milk of magnesia diluted with water ℥iv (125 cc.), with tincture of belladonna, ♏x (0.6), being poured into the stomach through the tube before removal. A hypodermic of morphin is then given, the room darkened, and the patient kept perfectly quiet. This treatment is repeated if necessary. If the patient will not consent to lavage, then alkalis, gelatin and whites of raw eggs with belladonna should be administered.

GASTROSUCCORRHEA (CONTINUA CHRONICA)

(*Synonyms.*—Chronic Gastrosuccorrhea; Chronic Hypersecretion; Chronic Parasecretion; Chronic Continuous Secretion of Gastric Juice; Reichmann's Disease.)

Chronic continuous secretion of the gastric juice is a perversion of function which is characterized by the secretion of an excessive quantity of gastric juice, not only after the ingestion of food but also when *the stomach is empty*.

The chief diagnostic point is the secretion of large quantities of gastric juice in the fasting stomach. Reichmann,[1] in 1882, was the first to describe this condition.

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[1] Berlin. klin. Wochenschr., 1882, No. 40.

We must make a distinction between the pure *cases of chronic* gastrosuccorrhea and those in which dilatation (atonic) of the stomach or dilatation from stenosis, especially with ulcer, exist, and with which hypersecretion is an associated symptom.

After fasting, small quantities of gastric juice, as much as 5 to 10 cc. or 15 to 20 cc., on one or two occasions have been aspirated. Thus the possibility of a normal small secretion may exist. If, however, we find 20 to 30 cc. or more of gastric juice constantly in the stomach of a patient who has been fasting for some time, the finding we would consider to be pathologic and to be an evidence of continuous hypersecretion.

Frequency.—Cases of pure gastrosuccorrhea chronica without any associated organic lesions of the stomach I believe, with Einhorn, to be quite a rare disease, while those found in association with ectasia are not uncommon. Undoubtedly the latter class have been confused with the pure cases. Chronic hypersecretion is more rare than the periodic type.

Etiology.—Chronic hypersecretion occurs more frequently in men than women, and in youth and middle life.

Severe mental strain or worry seem to be factors, also the neurotic condition.

The periodic type may develop into the chronic; persistent hyperchlorhydria or its causes, such as indigestible or irritating food or condiments, the abuse of alcohol or tobacco, or excessive hot or cold food and drinks, may be factors.

Dilatation of the stomach, ulcer, or tetany may have chronic gastrosuccorrhea associated with them.

Symptoms.—In most of the cases the patients state that the gastric symptoms began gradually, a feeling of pressure, fulness, and sour eructation commencing a couple of hours after the ingestion of food, resembling the symptoms of hyperchlorhydria. Then pain occurs several hours after meals or shortly before the ensuing meal; it is *most frequent on the empty stomach*, just before the next meal is due. It may be spasmodic in character; nausea and then vomiting follows. The vomiting may increase in frequency and, finally, occur several times a day after breakfast and supper. In some cases the attacks take place during the night between 12 and 2 o'clock, the patient being awakened by burning and pain in the epigastrium, acid eructations, and, finally, vomiting of very acid fluid takes place; after vomiting the pain is relieved. The night vomitus is generally a clear fluid. Albuminous food, egg or milk, will often relieve the pain. The day vomitus is very acid (quite liquid), but some food is mixed with it; is often of a grass-green color.

The appetite is generally good and often increased. Sometimes the patient develops excessive hunger; loss of appetite is rather rare.

Thirst is increased, the bowels are constipated, the urine diminished and less acid than normal.

There may be some loss of weight, but no marked emaciation.

The contrast between the physical condition of the pure type of chronic hypersecretion and that in which dilatation of the stomach accompanies it will be described under Differential Diagnosis.

Palpation.—The stomach is more or less sensitive to pressure.

Diagnosis.—The diagnosis of gastrosuccorrhea continua chronica can only be made *by examination of the stomach in the fasting condition.* To exclude error, the method suggested by Reichmann is the best.

The stomach should be thoroughly washed out with warm water, preferably at night, all food and drink should be withheld for ten to twelve hours, and then aspiration of the stomach contents should be performed. Anywhere from 50 to 125 cc. of gastric contents is thus obtained. This secretion exhibits all the properties of the gastric juice. It contains no particles of food. The fluid is usually watery and clear in color or it may be yellowish green from admixture with bile. There is an increased degree of acidity, the values for free hydrochloric acid being, as a rule, pretty high. There are no starchy products contained therein. There are no evidences of fermentation and no organic acids present. Under the microscope no sarcinæ are found.

An hour after Ewald's test breakfast more liquid than usual is present and the acidity is quite high (75 to 125), higher than when the fasting contents are aspirated. A thin disk of egg-albumen will be digested at blood temperature in an hour or so. Lugol's solution gives a blue or violet reaction, showing the starch is unchanged or slightly so.

Three to four hours after Riegel's test dinner hardly any meat fibers are found, whereas considerable starchy material is present. The acid content is high and the residue considerable.

We must remember that the above description is a case of pure chronic gastrosuccorrhea, in which there is no motor insufficiency. These cases I believe to be quite rare.

Differential Diagnosis.—Many of our writers do not properly distinguish between cases of pure chronic hypersecretion and those in which dilatation of the stomach is a complication. The examination of the stomach as regards its size, position, and motor functions should always be carried out, and this will aid us.

Ulcer of the stomach may be complicated by hypersecretion, and in a few rare cases gastric tetany is associated with it. Occasionally no symptoms pointing to a previous ulcer can be obtained, though there may have been a previous history of hyperchlorhydria, and at subsequent operation the stenosis will be found to be due to an ulcer not entirely healed.

In *ectasy* from pyloric benign stenosis with hypersecretion we have excessive vomiting of a large quantity of greenish-yellow fluid, pain, cramp-like attacks, peristaltic unrest, excessive thirst, marked loss of weight, skin over the abdomen dry and wrinkled, loss of weight which is often very great, and occasionally some tinge of blood in the vomitus. The patient presents almost a cachectic appearance, and there is in some patients the sense of resistance, or even a feeling of thickening at the pylorus.

The vomitus or contents aspirated after a test meal will separate into three layers: an upper layer of foam, middle layer yellow or yellowish green, and a lower layer of sediment. Meat is digested, the sediment consists of starchy material. Acidity is markedly increased and content of hydrochloric acid is high. Pepsin digestion is rapid. Starchy materials, yeast-cells, and sarcinæ are found under the microscope.

Examination further shows marked dilatation of the stomach, with great relative motor insufficiency. *Hyperchlorhydria with atonic dilatation must also be differentiated.* The pain and vomiting present some of the symptoms of hypersecretion.

For example, in one case a test meal given at night shows A. M. on aspiration 500 cc. of contents; separating into three layers, fermentation being present, stomach one finger below umbilicus. Reichmann's method was then employed, *i. e.*, the stomach was thoroughly washed out and nothing given for twelve hours. Morning aspiration showed the organ to be empty, no secretion, hence the diagnosis was clear.

In many cases of marked stagnation of the stomach contents with dilatation, continuous secretion may be simulated. The method of testing the empty stomach will determine the diagnosis.

If there have been hematemesis, melena, circumscribed tenderness, or dorsal tenderness ulcer will be suspected. Frequent examinations for occult blood in the stomach contents and stool are of service.

Tetany may complicate hypersecretion with dilatation. The symptoms are characteristic.

Prognosis.—In the cases of pure chronic hypersecretion the prognosis is fairly good. Most patients improve under treatment, but quite frequently there are relapses. Sometimes the condition persists for years. Hypersecretion *per se* is never fatal. When complications such as dilatation of the stomach are present the serious features are dependent on them.

Treatment.—*Prophylaxis.*—The patient should not overwork, should be relieved of all mental oxerexertion, and lead a rational out-of-door life, with proper attention to exercise and hygiene. Nervous conditions when present should be treated. The patient should avoid bolting his food, should not eat any irritating substances, such as mustard, pepper, spices, alcohol, and very hot and very cold food and drink; in fact, he should avoid everything that will overstimulate the secretion of gastric juice.

Albuminous food is digested well, and starchy food, badly; hence the latter should be reduced in quantity.

Diet.—This is practically the same as in hyperchlorhydria, except that very large quantities of fluid should be avoided.

If the appetite and physical condition are good, it is just as well to give but three meals a day, so as to give the stomach a rest and not tend to keep up gastric secretion. In this event the excessive acidity can be neutralized between feedings by alkalis. Doses, ʒss to j (2.0–4.0) of magnesia usta, milk of magnesia, or soda bicarb. should be given, as in hyperchlorhydria, in water one to two hours after meals, and the hypersecretion and the pain relieved by tincture of belladonna, ♏x (0.6), t. i. d.

On the other hand, some patients, as in hyperchlorhydria, readily feel satiated, and yet desire food frequently. They may also be losing some weight, especially in the cases complicated with dilatation; small meals, which are readily expelled from the stomach (*i. e.*, in soluble form or mushes), must be given, and yet the nutrition must be kept up, which last necessitates frequent feeding.

The diet, for example, when given in small frequent meals would be as in hyperchlorhydria, but *less fluid:*

7.30 A. M.—Milk or cocoa, 250 cc. (℥viij), 2 zwieback or toast and egg (1).
10.00 A. M.—Beef sandwich or ham sandwich.
1.30 P. M.—Soup, 250 cc. (℥viij), with raw egg, steak (gm. 100), potatoes (gm. 50).
4.00 P. M.—Same as 10 A. M.
7.00 P. M.—2 eggs or meat (gr. 100), 2 slices toast, butter (gm. 20).

If there is dilatation, more soluble food should be given (see Dilatation of the Stomach). Starches should be given thoroughly cooked, or predigested and in small amounts, and preferably in soups and mushes; potatoes should be mashed and alcohol avoided; also avoid cabbage, turnips, spices, pickles, mustard, etc.

Medicaments.—*To lessen hypersecretion.*

Tinct. belladonna in doses of ♏x (0.6) or more, t. i. d. before meals, up to physiologic effects, or ext. belladonna, gr. $\frac{1}{6}$ to $\frac{1}{3}$ (.01–.02), will lessen secretion and subsequent hypersecretion. Atropin, gr. $\frac{1}{100}$ to $\frac{1}{50}$ (0.0006–0.0013) t. i. d., by mouth or hypodermic, is also of value; the pain and the spasm of the pylorus and the hypersecretion are lessened by these remedies.

One can administer a large dose of belladonna after lavage before withdrawing the tube. Bismuth subnitrate, gr. 30 (2.0) in ℥ij (60.0) water, t. i. d. half an hour before meals, or olive oil, ℥j (30.0), t. i. d. before meals, or the latter containing gr. 30 (2.0) of bismuth lessen secretion.

Large doses of morphin, as have been recommended, I believe to be a pernicious method for obvious reasons.

For Attacks of Pain.—Alkalis, such as milk of magnesia (Phillips), ℥ss (16.0) in ℥ij (60.0) of water, or magnesia usta, ʒj (4.0), or soda bicarb. alone, ʒj (4.0), or combined with the above, are of service.

Albumen-water (white of raw egg) or gelatin, 5 to 10 per cent. solution (j℥—30.0), are useful. Heat should be applied externally. *Lavage, preferably* with *an alkaline solution*, with ℥j (30.0) milk of magnesia in 1 quart (liter) of warm water, or with soda bicarb., ℥ss (16.0), or magnesia usta, ℥ss (16.0) in the same amount of water, is the best method. A small quantity may be left in the stomach and belladonna, ♏x (.06), with an additional dose of the alkali poured in before removing the tube.

In some cases it may be necessary to administer a hypodermic of codein, gr. ¼ to ½ (0.0016–0.032), or morphin, gr. ⅛ to ¼ (0.008–0.016). They should only be given by a nurse or physician.

Alkalis.—If there be no ectasy, a course at Carlsbad is of service, or artificial Carlsbad salts or the imported salts, or alkaline mineral waters, such as Vichy, can be taken at meals or just before meals. This lessens hyperacidity and thus aids digestion.

To prevent subsequent attacks of pain an alkali should be given at the height of digestion, about two to two and a half hours after food; magnesia usta or milk of magnesia, ʒss to ij (2.0–8.0), alone or combined with soda bicarb. or ammonia magnesia phosp., as described under Hyperchlorhydria, are useful.

Lavage.—Reichmann and Riegel were the first to recommend lavage for the treatment of this condition. Reichmann and Einhorn perform lavage in the morning with the stomach fasting, while Riegel washes it out six or seven hours after the heavy meal. My method is entirely dependent on the time of appearance of the symptoms. If the attack comes on at midnight or early in the morning, it seems most logical to perform thorough lavage at bedtime with an alkaline solution, leaving some of it in the organ, and also pouring into it a large dose of belladonna—♏x to xv (0.6–1.0).

If the attacks come on after breakfast or the noon meal, then the early morning lavage on the empty stomach seems best. I often recommend pouring Carlsbad salts directly through the tube before withdrawal. With *severe pain* it may be necessary to perform lavage again during the exacerbation.

If there be dilatation and marked retention of food, lavage six hours after the full noon meal is necessary, then followed by a light supper; an alkali and belladonna should be given at bedtime.

In some obstinate cases Reichmann recommends lavage with 1:1000 to 1:2000 silver nitrate solution. It is safest to wash the stomach with about 500 cc. of this solution, 150 cc. at a time, and then wash out the stomach with warm water. Normal salt solution may be substituted if there is much irritation. Some also recommend the internal administration of gr. ⅙ to ¼ (0.011–0.016) of silver nitrate in pure form or in solution t. i. d. and at bedtime.

Stomach Spray.—Einhorn claims excellent results from spraying the stomach with his gastric spray with nitrate of silver 1:2000 to 1:1000 after a previous washing with warm water.

Direct Galvanization.—The same author reports good results from internal galvanization of the stomach, employing it and the gastric spray on alternate days. Riegel finds no benefit from intragastric galvanization unless atony is present, and in this I agree.

If the condition of chronic hypersecretion be complicated by dilatation, this condition must be treated. Rose's belt is of value for atonic ectasia. If there is pyloric stenosis, gastro-enterostomy is indicated.

Lavage with an alkali, belladonna or atropin in large doses, the alkaline treatment, diet, and in some cases spraying or lavage with nitrate of silver, are the chief requirements.

CHAPTER XV

DISTURBANCES OF THE MOTOR FUNCTION OF THE STOMACH—ACUTE ATONY—CHRONIC ATONY—ACUTE DILATATION OF THE STOMACH—CHRONIC DILATATION OF THE STOMACH

ATONY OF THE STOMACH

ATONY of the stomach may be defined as a relaxation and weakening of the muscular wall of the organ, so that it cannot empty itself in the normal time, and thus motor insufficiency results. It is a perversion of motor function. With simple atony, the stomach is of *normal size*, but motor insufficiency exists. When the organ is enlarged, we speak of ectasy or dilatation, which is combined with motor insufficiency.

There are two types of atony of the stomach: First, *acute atony;* second, *chronic atony*.

ACUTE ATONY OF THE STOMACH

Acute atony of the stomach may occur as a preliminary to acute dilatation of the stomach, just as may chronic atony to chronic atonic dilatation.

Acute atony does not necessarily result in acute dilatation.

Many of the causes which produce acute dilatation are the factors with acute atony, but the stomach has *not become dilated*. The early recognition of the condition is, therefore, important. The motor insufficiency which occurs with it may also *lead to error in diagnosis*. The condition takes place most frequently after overloading the stomach, bolting the food, or indigestible food, or alcohol. It may complicate acute gastritis, with belching, fulness or discomfort in the stomach, some distention, constipation, and *delayed vomiting;* in fact, merely discomfort for a considerable period of time and then the *vomiting of food taken some hours before*. The splashing sound is present. The stomach becomes distended, tympanitic, and often sensitive to pressure. A sudden attack of retention of chyme for an abnormal length of time is the salient symptom.

In one case, because of motor insufficiency following a single dietary indiscretion, a diagnosis of ectasy with motor insufficiency was made. Examination demonstrated normal functions, the attack evidently being an acute atony (acute motor insufficiency) of temporary duration.

During typhoid or the infectious diseases, gastric disturbances with belching, discomfort, sudden *distention of the stomach*, constipation, or diarrhea, with *delayed vomiting*, the vomitus consisting of milk (curdled) or *other food taken some hours previously*, are significant of this condition. There may be evidences in *the stool* of nourishment *taken forty-eight hours before.*

Treatment.—The immediate emptying of the gastro-intestinal tract is the indication. *Lavage should be performed at once.*

A good cathartic, calomel, gr. 3 to 5 (0.2–0.3), or blue mass, gr. 5 (0.3), followed by a saline cathartic, should be given.

Castor oil, ℥j to ij (30.0–60.0), is also excellent. Enemata, and especially rectal irrigation, are of value to produce intestinal peristalsis.

Acute atony may progress rapidly to acute dilatation or develop more slowly for ten to twelve hours, and then, if untreated, result in acute ectasy. Its early recognition is, therefore, important.

CHRONIC ATONY OF THE STOMACH

(*Synonyms.*—Motor Insufficiency; Gastric Insufficiency; Motor Insufficiency of the First Degree; Myasthenia Ventriculi; Atonia Gastrica.[1])

Chronic atony of the stomach, if untreated, may result in the atonic type of dilatation of the stomach.

Its correction is, therefore, of great importance. We use the term "chronic" in distinction from the acute, evanescent form. In the *pure cases no dilatation is present.*

Etiology.—Atony of the stomach may complicate many digestive disorders, such as chronic gastritis, hyperchlorhydria, neurasthenia gastrica, and diseases of the heart and lungs, as tuberculosis. It occurs in nervous and hysteric subjects and may exist as a *primary neurosis.*

It may occur as a result of biliary colic or the crisis of tabes.

Symptoms.—If atony occurs as a complication of some other affection of the stomach, its symptoms will be overshadowed by the primary disease. The characteristic symptoms of atony are: a feeling of fulness after meals, slight distention, belching of gas, diminution of appetite, headache, and constipation. The resulting motor insufficiency is productive of fermentation and gas production.

Physical Examination.—There is generally some distention of the stomach with gas. The splashing sound is easily produced over the greater part of the stomach an hour or two after the test breakfast, or four or five hours after a full meal. If the splash extend to the *umbilicus* or *below it*, this is evidence that *dilatation* is associated.

If movable kidney is associated, gastroptosis is present. These are not pure cases.

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[1] Rose has called to our attention that atonia gastrica is an improper term, really meaning abdominal relaxation. It is so applied in our work to define splanchnoptosis.

An hour after Ewald's test breakfast aspiration of the stomach contents will remove 100 cc. or more of gastric contents; an excellent test taken in connection with the symptoms.

Six hours after Leube's test dinner aspiration and lavage show considerable chyme, 150 to 200 cc. The fasting stomach in the morning is found empty.

Boas states that on filling the stomach with water, the greater curvature will descend as water is added. This is not reliable. The lower border will descend after a moderate amount of water, if the stomach is dilated.

Prognosis.—This is good if proper treatment is instituted.

Treatment.—If hyperchlorhydria or chronic gastritis is present, each should receive appropriate treatment; as should tuberculosis, endocarditis, nervous conditions, etc.

In all cases of chronic atony of the stomach, Rose's plaster belt is indicated. Atony of the intestines is frequently associated, and the use of mechanic support lends tone to the general musculature of the abdomen, keeps the stomach well supported, and prevents *atonic dilatation of the organ.*

Vibratory massage or massage over the entire gastro-intestinal tract is of value. It stimulates the muscles and lessens the tendency to constipation. Outdoor exercise, walking, golf, and horseback riding are indicated. In some cases douching the abdomen is of service; in this event one must employ a silk elastic abdominal support, which can be readily removed. I have secured the best results with Rose's belt. The patient should not overwork, either mentally or physically, and should eat slowly and masticate the food thoroughly; the teeth should be kept in good condition. An excessive quantity of fluid should not be taken—in all, including water, soups, tea, etc., not over 1½ liters a day.

It is best to give numerous divided meals of rather moderate size, four or even five daily; so as not to overburden the stomach with three large meals, and allow it to thoroughly empty itself.

The diet may include bread (fresh and hot breads are interdicted) and butter, eggs in various forms, cereals, milk, soup, chicken, steak, chops, game, squab, fish, oysters, and green vegetables, which are specially valuable for the constipation; cocoa, weak tea, occasionally weak coffee, with milk and sugar. The diet must be modified if hyperchlorhydria or chronic gastritis is present, or to suit the special idiosyncrasies of the patient. Alcohol should be interdicted.

Medicaments.—Strychnin or nux vomica is of great service to tone up the muscular system. They may be given alone, or

℞. Tr. nucis vomicæ.............................♏x (0.59 cc.)
Comp. tinct. cinchona..........................♏xv (0.888 cc.).—M.

Sig.—Give in a wineglassful of water t. i. d. half an hour before meals. Some prefer it at the same time after meals.

or, if the patient is anemic, it may be combined with iron and arsenic thus:

℞. Strych. sulph................................gr. $\frac{1}{30}$ (0.0021)
Sod. arsen.................................gr. $\frac{1}{50}$ (0.0013)
Blaud's iron pill..........................gr. v (0.3).—M.
One pill; administer t. i. d. after meals.

Strych. sulph., gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.021), is excellent, or tinct nux vomicæ in combination with fluidextract of condurango, or comp tincture of cinchona, thus:

℞. Tinct. nucis vomicæ,
Fluidext. condurango..................āā 125 cc. (about ℥ij).—M
Dose, 20 drops in water, t. i. d. before meals.

If hyperchlorhydria is present, I omit strychnin; though Musse advocates nux in the neurotic type.

Other useful iron preparations are:

Iron tropon...............................ʒj to ij (4.0–8.0), t. i. d.
Peptomangan (Gude)......................ʒj to iij (4.0–12.0), t. i. d
To be given in water or milk after meals.

Electricity.—This is of service applied by the external method o in some cases, by intragastric faradization.

Static electricity or the high-frequency current may be useful i nervous cases.

Lavage is not indicated.

Massage or *vibratory massage* is especially useful.

For Constipation.—The green vegetables, brown, rye, and Grahan bread, and raw or stewed fruits are serviceable. A glass of wate should be taken on rising. The patient should accustom himself t go to stool at a definite hour, and on the closet may gently massag the stomach and bowels to aid action.

A small gluten or glycerin suppository, or the injection of 1 ounc (30.0) of olive oil, or 4 to 6 ounces (125–185 cc.) of warm water hav a good effect in exciting peristalsis; or a soapsuds enema, but never o larger size than 1 liter (quart). Olive oil injection, 1 pint to 1 quar (500 cc. to 1 liter), at bedtime, to be retained, is useful.

It may be necessary to employ medication, such as extract o cascara, gr. 1 to 4 (0.065–0.26), at bedtime, or fluidextract of cascara ʒj to ij (4.0–8.0), or pills, such as the lapactic, aloin, belladonna podophyllin, phenolax, purgen tablets, etc.

ACUTE DILATATION OF THE STOMACH

Acute dilatation of the stomach may be defined as acute aton of the stomach, with resulting acute motor insufficiency, graduall merging into a paralytic condition, and accompanied by a distentio of the organ to beyond its normal physiologic limits. Its lowe border extends to the umbilicus or usually to below this point, and the stomach may even occupy the entire abdominal cavity.

Brunton, Fagge, Boas, Hemmeter, notably Campbell Thomson,[1] and Lewis A. Conner,[2] have written on this subject. The fatal or most severe cases have been reported, but the condition occurs quite frequently. I have already referred to numerous types.[3] Personal investigation demonstrates that five anatomic types of acute ectasy exist:

1. Acute dilatation of the stomach alone.

2. Acute ectasy, which supervenes on an existing chronic dilatation (due to stenosis of the pylorus). Thomson also reports one case.

3. Acute dilatation of the stomach and duodenum, the most fatal type.

4. Acute dilatation of the stomach engrafted on chronic atonic dilatation.

5. Acute dilatation of the stomach and intestines, a mixed type, so-called acute tympanites.

This last is quite common, especially in the acute infectious diseases, such as typhoid fever and pneumonia.

For a complete description of the theories of its mechanism and of the subject, I refer my readers to the "American Journal of Surgery," November–December, 1908. Weston and the author found that the fibrous attachment of the transverse duodenum to the diaphragm (muscle of Treitz) and the pressure of the dilated stomach on the transverse duodenum were chief factors in the production of the gastroduodenal type of dilatation. Mesenteric traction was chiefly produced by the downward pressure of the stomach on the intestines, which last exercised a countertraction against the muscle of Treitz. The collapsed intestines were the result of pressure.

Mechanism of the Production of Acute Dilatation of the Stomach.—The nature of the condition as stated is undoubtedly an *acute atony*, with acute *motor insufficiency*, finally merging *into a paralytic condition*. Many factors in the production of acute dilatation have been described, namely:

1. Section of the vagi—by Carion and Hallion—producing acute dilatation of the stomach, thus demonstrating that an injury or inflammation of these nerves may be a cause, as in cerebral injury, or pneumonia at the base.

2. Injury to the dorsal spine by stimulation of the inhibitory nerves.

3. Direct action of the agent on the musculature or its terminal nerve filaments; among such may be chloroform, anesthesia, toxemia from fermentation, etc.

4. Traumatism.

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[1] Brochure.

[2] Am. Jour. of the Med. Sci., March, 1907.

[3] Medical News, August 6, 1904.

5. Spasmodic stenosis of the pylorus, due to fermentation or h peracidity.

6. Acute gastrorrhea (Morris); or possibly acute gastrosuccorrhe (Kemp).

7. Kelling's and Conner's experiments on spasmodic closure c the cardia, and the demonstration of kinks in various parts of th duodenum, or of spasm of the pylorus.

8. Rotation of the pylorus.

9. Kelling's and Braun's experiments demonstrating that acut dilatation of the stomach is a paralytic condition, by producing with animals under deep narcosis.

10. Rotation at junction of cardia and esophagus.

11. Toxemia from infection, as from the toxins of typhoid, pne monia, etc,; or auto-intoxication from improper diet, causing gastr intestinal dilatation during the course of these diseases. Dietary i discretions are the causes of acute ectasy of the milder types which shall shortly describe.

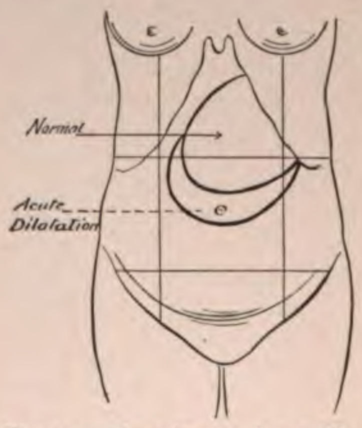

Fig. 140.—Experiment: Acute dilatation by CO_2 distention.

12. *Obstruction of the transver duodenum* is one of the freque causes so far found in the fatal case producing acute gastroduodenal d latation, the most dangerous typ There are a number of causes giv for this condition, notably:

(*a*) Mesenteric obstruction the duodenum from mesenteric tra tion. Out of 69 fatal cases 19, 27.5 per cent., Conner states we caused by this, and probably to 50 per cent. is nearer the fi ure, according to his view. A brecht first called attention this condition, performing nume ous experiments.

(*b*) Pressure from the distended stomach on the transverse du denum, producing complete obstruction, is most frequently the cau in my belief.

(*c*) The firm fibrous band (muscle of Treitz) attaching the tran verse duodenum to the crus of the diaphragm, a factor hereafter note and demonstrated by Dr. Weston and myself, is also an importa factor.

Regarding mesenteric traction, Conner further believed that t conditions essential for this were the dorsal position, an empty inte tine, and a mesentery of such length that the intestine can slip in the pelvis and yet hang free. He believes fasting, purges, and enem after operation have a possible bearing.

Accessory factors are suggested, such as a lax abdominal wa pressure from weight of hepatized lungs (in pneumonia), coughing and laughing paroxysms, lordosis, or an abnormal position of the duodenum. It generally crosses the third lumbar vertebra; the fourth is the most prominent, and more pressure would be exercised at this point.

Clotted blood behind the transverse duodenum was a cause in one case.

On the other hand, some believe the stomach dilatation is the primary factor and the mesenteric constriction is produced secondarily by the stomach forcing down the intestines.

T. Satterthwaite and the author studied the effects of acute gastric distention on the pulse and respiration by artificially distending the stomachs of patients with carbonic acid gas and taking the blood-pressure before, during, and after the experiment. In Fig. 140 is illustrated the result in one of our cases.

Physician....................Position dorsal.

	Pulse.	Pressure.	Respiration.	Pulse.
Before distention.........	72	135	18	Slightly irritable (tobacco).
After distention..........	86	120	24	Irregular in force; some intermission in the beats.

Pain under the sternum and over the abdomen, sensation of suffocation and of flushing of the face, nausea, and discomfort accompany the acute distention. The stomach extends to one finger-breadth below the umbilicus; in all a distention of $3\frac{1}{2}$ fingers-breadth.

Aspiration of the contents relieved the symptoms. These facts emphasize the danger of overdistention of the stomach with carbonic acid gas for testing the position of the organ in patients with cardiac or pulmonary disease or in old age; also the danger of acute ectasy as a complication. Moreover, the true dimensions of the stomach may not be obtained by the method of carbonic acid gas distention.

Etiology of Acute Ectasy.—It may be primary or secondary. The causes are as follows: Indigestible food; infectious diseases, such as typhoid, pneumonia, acute tuberculosis, and scarlatina; during convalescence from long-continued disease, as chronic tuberculosis, hip-disease, pneumonia, typhoid, sarcoma, and anemia; injury to the head or spine; traumatism to the abdomen; postoperative, in which manipulation of the viscera, shock, uremia, sepsis, and the anesthetic are factors; one case after gastro-enterostomy reported by the author; retroperitoneal abscess; disease and deformity of the spine, lordosis, etc.; application of plaster jacket in spinal deformity; paroxysm of laughing supposedly (true cause undiscovered). Toxemia or auto-intoxication are, therefore, factors in many cases.

Age.—It may occur from infancy to old age. Three-fourth: the cases are developed during adolescence or early adult life (te forty years).

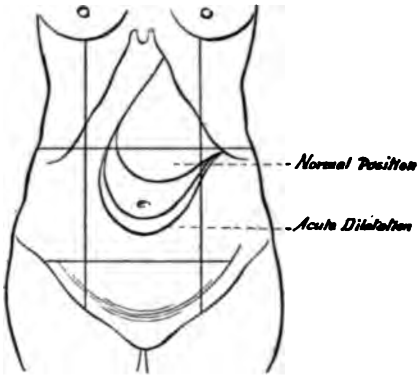

Fig. 141.—Acute dilatation of the stomach during epileptic attack.

Sex.—Is about equally divided.

Clinically, we may classify acute dilatation of the stomach i cases presenting various clinical types, in this sense atypic, and i the typic cases, which are usually described.

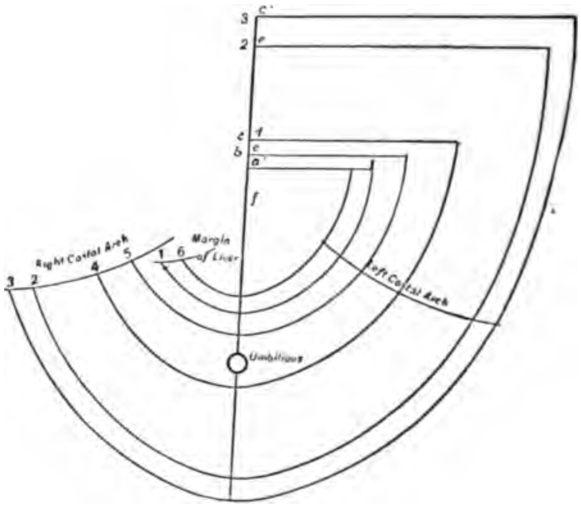

Fig. 142.—Acute dilatation of the stomach in migraine, August 29, 1 Borders of stomach: 1, August 16; 2, August 29, in the morning; 3, August in the evening; 4, August 30, in the morning; 5, August 30, in the eveni 6, August 31, in the morning.

Clinical Types of Acute Ectasy.—I shall briefly refer to th cases, which have already been fully reported by me.[1]

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[1] American Journal of Surgery, Nov.–Dec., 1908.

1. *Cases of Acute Dilatation of the Stomach with Symptoms Pointing to the Nervous System.—Convulsions in Infants and Young Children.*—Auto-intoxication is the cause. I have seen a case in an infant two years of age in which the stomach extended 2 inches below the umbilicus. Vomiting of bread and curds occurred, with immediate cessation of convulsions and return of the stomach to normal position. Repeated attacks may lead to chronic ectasy or epilepsy.

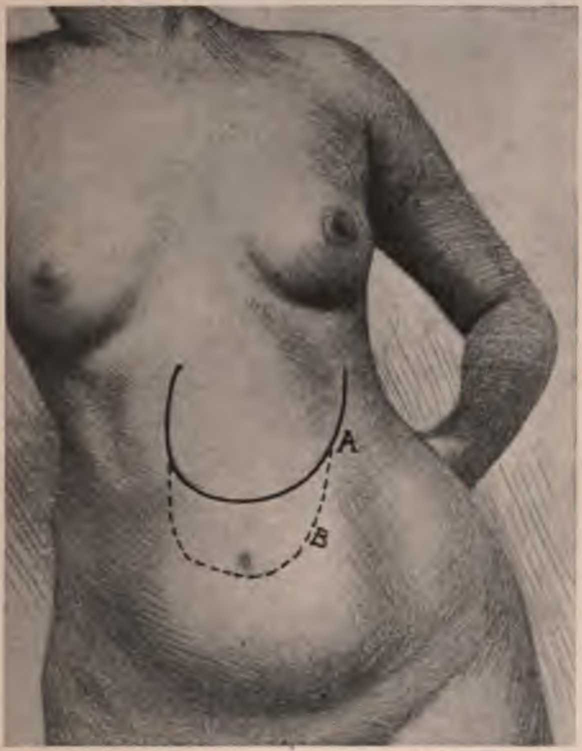

Fig. 143.—Acute dilatation of stomach with tachycardia.

Epilepsy.—Mangelsdorf[1] has demonstrated acute ectasy during the convulsive seizure and the gradual return to normal position (Fig. 141).

Migraine.—The same author noted similar conditions during attacks of migraine (Fig. 142), and reports *500 cases in epilepsy and migraine.*

Lauder Brunton[2] noted transitory dilatation in sick headache.

Tetany.—Broadbent[3] describes a case of *acute ectasy with tetany,* which ended in recovery.

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[1] Atonia Gastrica, Rose and Kemp.
[2] Allbutt's System of Medicine, vol. iii, p. 392.
[3] Practitioner, 1908.

Chorea.—Acute ectasy has been reported as a terminal event in chorea.[1]

3. *Acute Ectasy Producing Acute Cardiac Symptoms.—Tachycardia.*—Girl, age twenty-one, with chronic endocarditis, excellent compensation, no gastric disturbances. Tachycardia, 210 beats per minute, followed dietary indiscretion. Acute ectasy was found as in Fig. 143. Emesis occurred, the stomach contracted to normal size, and the tachycardia ceased. The patient suffered from several attacks, but has had no further trouble since she has exercised care in diet.

Pseudo-angina Pectoris.—Female, age sixty-five, suffered from attacks of pseudo-angina, following dietary indiscretions, at times consciousness was lost. A number of attacks occurred and in every instance acute ectasy was present, as in Fig. 144. Emesis relieved both the dilatation and the attack. Ultimate cure resulted from proper diet.

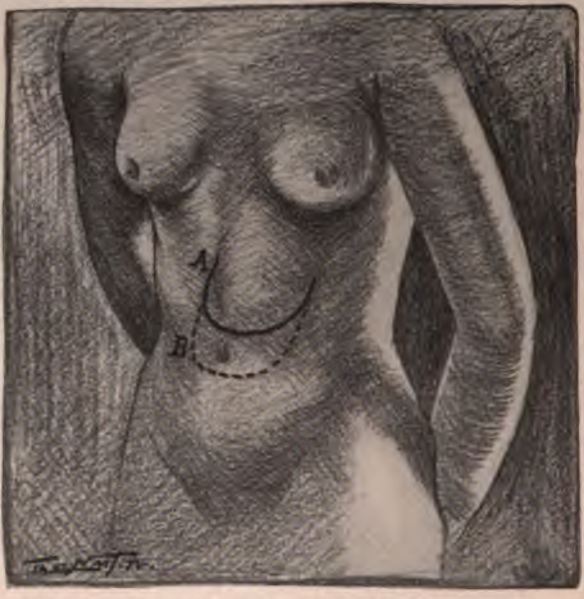

Fig. 144.—Acute dilatation of the stomach with symptoms of pseudo-angina pectoris.

4. *Acute Ectasy Complicating Infectious Diseases, Notably Typhoid and Pneumonia.*—The tympanites of typhoid is frequently not purely intestinal. Acute gastro-intestinal dilatation is by no means rare; in fact, I have found it quite frequent. Systemic infection or improper diet cause this condition. Acute gastroduodenal dilatation has been reported.

In the milder types of acute gastro-intestinal dilatation there is often *no vomiting.* There are cardiorespiratory symptoms which might suggest pulmonary involvement. Examination shows acute distention. Postural treatment,[1] by elevation of the head of the bed, enteroclysis, and lavage, will relieve the symptoms. The same mixed type of distention may occur with pain and shock and simulate perforation. After relief of the distention, examination shows *absence of muscular rigidity* (no peritonitis).

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[1] Lancet, April 19, 1890.

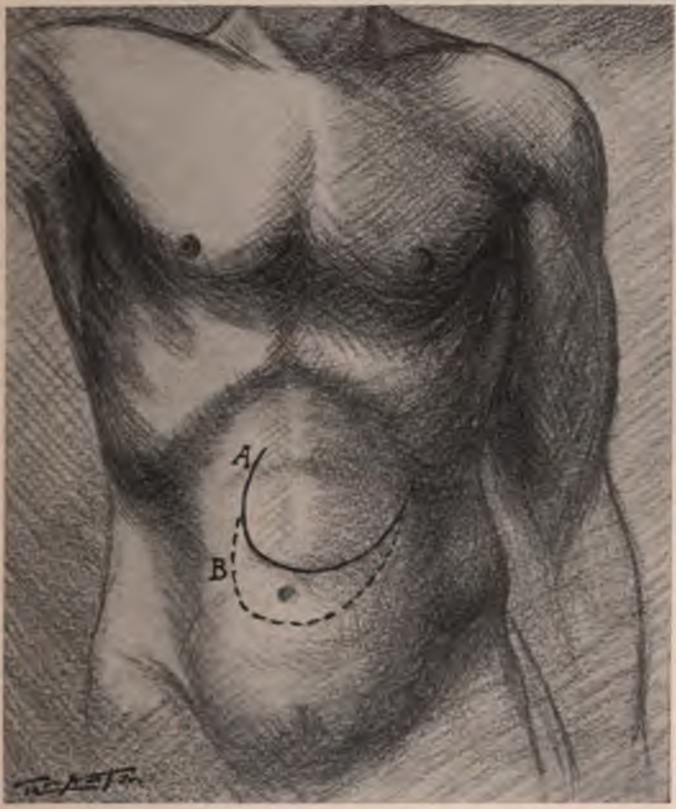

Fig. 145.—Acute dilatation of the stomach, with constipation (ten days' duration) a prominent symptom.

In the acute distention of typhoid, with intestinal hemorrhage, lavage will relieve gastric distention and diminish intra-abdominal tension. I have never seen it recommended.

Pneumonia.—We may have the gastroduodenal type of acute ectasy, of which several cases are reported. The mixed type is quite common and constitutes a serious danger. Undoubtedly sudden heart-failure has been precipitated by this condition. The etiology and treatment are the same as in typhoid fever. There is greater danger to the heart from the pressure than from the passage of the stomach-tube.

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[1] See Treatment of Typhoid Fever.

5. *Acute Ectasy with Coprostasis the Prominent Symptom.*—Male, age fifty-five, following indiscretions in diet, complained chiefly of coprostasis, with occasional vomiting. Constipation had been present eight days when I saw the case. Fecal impaction was present in the sigmoid and caput coli.

Frequent lavage, enteroclysis, and cathartics relieved the condition. In Fig. 145 is depicted the stomach before and after relief.

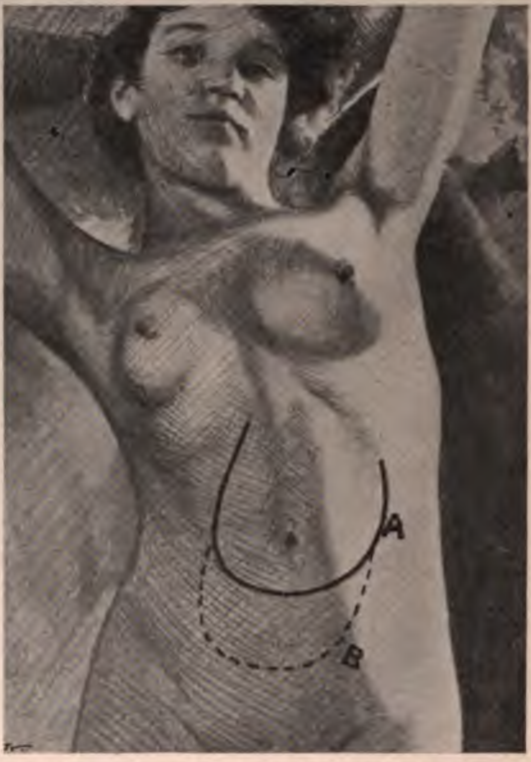

Fig. 146.—Acute dilatation of the stomach engrafted on chronic dilatation.

6. *Acute Dilatation Supervening on Chronic Ectasy.*—Female, age forty-five, suffered from chronic ectasy due to pyloric stenosis from ulcer. Following a dietary indiscretion, acute ectasy resulted with gastrosuccorrhea, pain, vomiting, peristaltic waves, constipation, and collapse.

In Fig. 146 is shown the acute ectasy, with return to the position of chronic ectasy after lavage. Subsequently gastro-enterostomy was performed with a gain of 100 pounds in the patient's weight.

Symptoms of the Severe Cases.—The symptoms are characteristic, and are as follows:

Sudden abdominal distention, pain, tenderness, excessive vomiting, constipation, thirst, scanty urine, and collapse. It has been mistaken for intestinal obstruction or for peritonitis.

Onset.—This is nearly always sudden. The patient may be well or suffer from some illness taking its usual course, when he suddenly complains of great distention, discomfort, or severe pain in the abdomen. This is rapidly followed by vomiting, which is the most constant symptom, begins early, and generally persists throughout the attack. Rarely there may be an intermission due to temporary cessation of secretion, or cessation of vomiting may be a terminal event, the abdominal muscles and diaphragm being no longer able to expel the contents. Cessation of vomiting is, therefore, not always a favorable symptom.

The vomiting is profuse, in large amounts, and comes up in gulps without straining. In the early stage it may consist of the gastric contents, of food in various degrees of fermentation; later, it becomes thinner and watery, and generally of a greenish hue. It is often described as bilious. It may be brownish, grayish, or even inky black; occasionally there may be a trace of blood. Often in the postoperative cases acute distention, pain, and greenish vomiting are the first symptoms. The vomitus may be sour or foul or even (rarely) feculent in odor.

Character of the Vomitus.—Various constituents, such as bile, diastase ferment, hydrochloric acid, lactic acid and traces of blood, visible or occult, have been found.

Pain is present in the majority of cases, usually in the epigastric and umbilical regions. In the gastro-intestinal mixed cases at the commencement it is more general and acute from sudden distention, being suggestive of peritonitis[1]; later there is a feeling of distention, not so acute, with accompanying cardiorespiratory symptoms. It differs from the continuous pain of acute obstruction.

Tenderness occurs in some cases. *Muscular rigidity is absent.* The urine becomes scanty and nearly suppressed during the last twenty-four hours. Anuria is diagnostic of obstruction high up in the intestinal tract, and does not occur with obstruction of the large intestine. It has been mistaken for uremia. The temperature is usually normal or subnormal, unless the patient has fever. Thirst is marked. Hiccough may occur as a terminal symptom, as may also delirium. General muscular cramps occurred in one case; and Broadbent reports a case of tetany.

Physical Signs.—In the gastric or the gastroduodenal type there is distention of the abdomen, but the swelling is not uniform; it chiefly fills the left half and lower part of the abdomen, and the right hypochondrium appears to be flattened. There is often swelling in the epigastrium.

The following is of service: Draw a line from the tip of the ensiform to the junction of the middle and outer third of Poupart's ligament (Fig. 147). The distention usually lies to the left and below this oblique line. Occasionally it appears more below the navel and sometimes there is general distention.

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[1] Sudden perforation may even be suspected.

Splashing sounds (*succussion*) and the sense of fluctuation are an aid in some cases. They are not always present in the early period, when there is chiefly gas in the organ. They occur below the level of the umbilicus.

Percussion will show the resonance increased, but will be interfered with when there is much fluid. It is important when the splash is absent.

Peristaltic waves of contraction occur very seldom. They are found only before complete paresis takes place, or in the acute cases engrafted on the stenotic type of chronic dilatation.

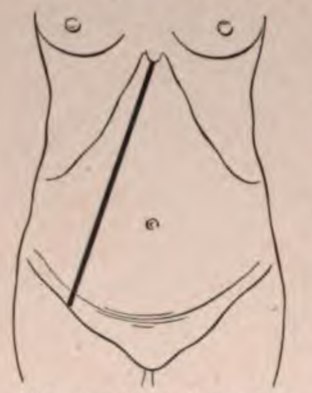

Fig. 147.—Line drawn from ensiform to Poupart's ligament.

The *general symptoms* are those of collapse, a rapid and small pulse, frequent respiration, a clammy skin, and subnormal temperature.

Duration of the Attacks.—The duration of the attack depends on its severity and type of case. In my case of tachycardia it lasted less than an hour. In the mixed cases it depends on the treatment accorded by the physician.

Among the *severe cases*, 1 case of Conner's died within three hours after the onset of the pain, which, with distention, was the first symptom. There was no vomiting. Several cases died within twenty-four hours, and sixteen days was the longest duration. The average was about five days. Some cases recur or several weeks may elapse before the dilatation entirely disappears.

Prognosis.—In the severe cases it has been extremely bad, being a most formidable condition. There is a 72 per cent. death-rate recorded. In reality many cases occur, and with our present knowledge the mortality should be comparatively small.

Morbid Anatomy.—The post-mortem appearance of the stomach is quite characteristic, being cylindric and bent into a horseshoe or V-shape. The cardiac portion is the longer (Fig. 148).

The walls of the stomach are distended and thinned. A large amount of elasticity is retained, as shown by the shrinking that takes place after the distending force is removed.[1] The stomach may occupy the entire abdomen or even reach into the pelvis. It has been mistaken on operation for cyst of the pancreas. Its color may be purplish red, grayish, or bluish white.

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[1] McEvitt, New York State Journal of Medicine, July, 1906.

Microscopic Examination.—This has been made in a few cases. There was thinning of the musculature and no definite microscopic changes were noted, though in some there were small hemorrhages.

Duodenum.—In a large number of cases—over 50 per cent. (Conner)—a part or the whole of the duodenum shared in the dilatation; in many the distention stopping where the mesentery crossed. Kinks have also been found in the duodenum. The coils of the intestines are flattened and collapsed in the pelvis.

Diagnosis.—One should always think of the possibility that tachycardia, convulsions, epilepsy, migraine, anginoid symptoms, increased cardiac and respiratory rapidity, and even tetany may occur with acute ectasy. Sudden gastro-intestinal dilatation is quite frequent in typhoid and pneumonia. Often vomiting is absent in the milder cases, and the chief symptom pointing to the abdomen is distention. At times there may be acute pain in typhoid, simulating perforation. Examination in every case should be made *by percussion, the splashing sound, and especially by lavage, if there is any doubt.*

Intestinal irrigation (recurrent) will often relieve distention when present, and aid in diagnosis.

Postnarcotic vomiting may become gradually persistent, and this should be looked on with suspicion.

Pain, tenderness, distention, continuous vomiting of bilious appearance are suggestive, especially with collapse, rapid and feeble pulse, *urinary suppression*, and obstinate constipation.

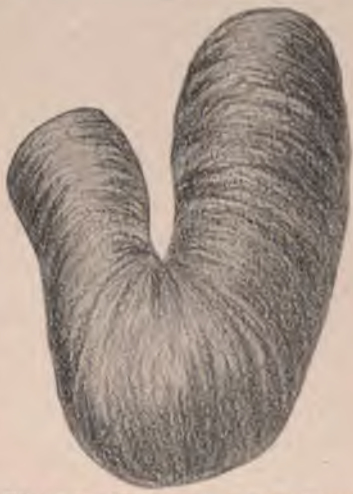

Fig. 148.—Post-mortem appearance of acute dilatation of the stomach.

In the severe cases there may be occasionally no vomiting and no pain, but only acute distention and some diarrhea. The use of the stomach-tube and determining the position of the *stomach before and after its employment* are of value in all cases.

If, after lavage, the distention disappears and there is no distention of the intestines or only slight tympanites in the colon, the acute dilatation is of the stomach or of the gastroduodenal type.

If intestinal distention persists, the dilatation is of the mixed type.

If all the tympanites is relieved by enteroclysis, subsequent examination will determine the stomach not to be dilated.

The presence of pancreatic juice, absence of fecal vomiting, and presence of bile show the gastroduodenal type. This is especially true if the abdominal posture affords relief. Bile, however, may be present with acute gastric distention alone.

True stercoral vomiting shows obstruction lower down, and *muscular rigidity* is a symptom of peritonitis.

Treatment.—*Prophylaxis.*—Rapidity in operating, the minimum amount of manipulation of the viscera, a minimum quantity of anesthesia, and care in feeding after operation are important.

The abolition of milk-diet in typhoid fever, pneumonia, and acute infectious diseases is advisable, with the substitution of broths, strained soups of various kinds, as barley, rice, and gruels. If the patient has had an attack, then the conditions of the gastric secretion should be investigated, irregularities corrected, and proper diet instituted.

In *every case* of acute ectasy the stomach should be immediately evacuated by lavage. It is an error to wait until the symptoms appear marked, or until the patient vomits, before lavage is instituted. The stomach may redistend in the severe cases.

It is advisable to repeat lavage within two hours or at times in three hours, and thereafter every four to six hours, during the first twenty-four hours, depending on the physical signs and symptoms. It may be necessary to perform it more frequently. At times it must be carried out for some days. It is safer *to err on the side of frequency.*

No food or drink should be given by mouth. For severe thirst, saline enemata, proctoclysis, or even hypodermoclysis may be administered. They are also efficient in the collapse, and infusion may be required. Rectal feeding must be kept up for several days until symptoms disappear.

If there is intestinal distention, continuous rectal irrigation is of value. It is advisable to promote peristalsis as soon as possible. Unless *hemorrhage*, peritonitis, or appendicitis complicate (as might occur in typhoid), *or there be a suspicion of a true intestinal obstruction*, after washing the stomach with plain water, in which milk of magnesia, 2 oz. (60.0), has been dissolved, I give calomel, gr. 3 to 5 (0.2–0.3), in water, ℥ss (16.0), directly through the stomach-tube before removal; and a saline cathartic by the same method four to six hours later.

In some cases I have given by preference a high enema[1] of ℥iv (125 cc.) of a saturated solution of magnesium sulphate two hours after lavage.

Tincture of belladonna is useful. It lessens the secretion, relaxes pyloric spasm, and has an excellent effect on the atony. It should be given in ♏v to x (0.296–0.592) doses on the tongue, with strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.00212), every four to six hours by hypodermic injection. The latter stimulates the musculature, the heart, and respiration. Atropin, gr. $\frac{1}{100}$ (0.00065), may be substituted hypodermically for belladonna.

Physostigmin sulph. (eserin), gr. $\frac{1}{100}$ (0.00065), has been recommended to promote evacuation of the bowel. I have recently employed gr. $\frac{1}{50}$ (0.0013) every two hours for several doses with success. It is well to arrange to give strychnin, gr. $\frac{1}{100}$ to $\frac{1}{60}$ (0.00065–0.00108), to guard the eserin.

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[1] This may be preferable, lest the patient vomit the saline cathartic.

Rectal electric irrigations of the bowels are efficacious for obstinate constipation.

The second most important therapeutic measure is *postural treatment.* The position depends on the anatomic type of the dilatation.

(1) The semi-oblique or nearly sitting position, *the head of the bed is blocked up* as in the illustration under Typhoid Fever. The patient lies on an inclined plane. This is of value in the *acute gastro-intestinal* (or mixed) *type*, with general abdominal distention, so frequent in typhoid or pneumonia, where the cardiac and respiratory symptoms are pronounced *from abdominal pressure.* By elevation of the head of the bed in one severe case of typhoid the tympanitic area in the thorax lowered 4 inches, and the pulse and respiration dropped 20 points each.

A fatal issue may result from pressure-effects on the heart and lungs with this type.

Frequent lavage and enteroclysis should be instituted in these cases, and later milk-free diet, substituting soups and broths.

This method would be *incorrect* in the gastroduodenal type of dilatation.

(2) Elevation of the foot of the bed to relieve pressure on the duodenum. The objectionable feature is *the danger from pressure on heart and lungs* if the stomach should begin to redilate.

(3) In the acute gastroduodenal type the *lateral position*, on the right or left side, has relieved the symptoms. The patient recovered.

(4) The abdominal position (*patient lying on the belly*) is *the best method* to treat the acute gastroduodenal type.

That this position affords relief seems to me to show quite conclusively that this type of obstruction is caused chiefly by the stomach pressure on the transverse duodenum.

Baumler kept the patient fifteen minutes in the knee-elbow position in each two hours; the balance of the time on the belly.

Operations.—These have not generally proved successful. Among those performed or suggested were:

The stomach opened and evacuated, and gastro-enterostomy. Gastric fistula might be tried. A kink at the duodenojejunal junction was relieved in one case, and the patient recovered.

Recovery has also been reported after one case of gastro-enterostomy.

Frequent lavage, combined with postural treatment, enteroclysis, and securing bowel action as soon as possible are indicated.

CHRONIC DILATATION OF THE STOMACH

(*Synonyms.*—Ectasy; Ectasia Ventriculi; Gastrectasy; Ischochymia—Einhorn; Motor Insufficiency of the Second Degree—Boas.)

The term "dilatation of the stomach" is employed for descriptive purposes, but in view of the existence of an acute type of dilatation of the organ, chronic dilatation of the stomach would seem a preferable nomenclature.

Definition.—How may dilatation of the stomach be defined? Is it to be measured by the capacity of the stomach alone or by the increased capacity plus the alteration of its functions? The latter is correct. The capacity of the normal stomach is extremely variable. Ziemssen has shown that a stomach may be normal and only contain 8 ounces (250 cc.), whereas another stomach, also normal, may possess a capacity of 2 quarts (liters). The large stomach ("megalogastria"), at times found during a physical examination, produces no symptoms. This may be congenital or acquired by large eaters or by those who live on vegetable diet. Such cases, however, can readily develop atony. As long, however, as the functions of the stomach are normal we cannot regard the conditions met with as pathologic, and hence cannot consider that dilatation exists.

As already described, the lower border of the normal stomach when distended with food or liquid lies from 1½ to 2 fingers-breadth above the level of the umbilicus.[1] If it descends to nearly the level of the umbilicus, to its level or below it, and symptoms accompany it, we must consider the organ dilated.

One must not commit the error of mistaking gastroptosis for dilatation. With gastroptosis the upper border of the stomach descends as well as the lower border, and there are movable kidney and enteroptosis.

The prolapsed stomach may, in addition, be dilated. There are varying degrees of gastroptosis. With dilatation the upper border of the stomach does not descend, but maintains its relation to the diaphragm, and the stomach is dilated chiefly in the direction to which the greatest force is applied, downward and laterally. The muscular fibers first elongate in the vertical direction and the distance between the lesser and the greater curvature is increased. Dilatation may also ensue in the transverse and anteroposterior dimensions and the pylorus may be a little further to the right and in a slightly lower plane, but the lesser curvature maintains its relation to the diaphragm, and this is the differential point between dilatation and gastroptosis (Fig. 149).

There is confusion as to the terms "atony," "ectasy," and "motor insufficiency," as they are often used interchangeably by different authors.

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[1] Examination of normal subjects, complaining of *no symptoms*, will frequently show that the stomachs are abnormally large, or in an abnormal position. Such cannot be considered pathologic.

Atony of the stomach may be defined, as already stated, as a loss of tone or contractile power of the muscles of the stomach, so that the organ does not contract about its contents, with a resulting *motor insufficiency* (*inability to expel its contents within the normal limit of time*). This condition has not progressed to dilatation.

Ectasy may be defined as dilatation, an enlargement of the stomach permanent in character, combined with motor insufficiency. With the atonic type of ectasia, there is motor insufficiency.

Relative Motor Insufficiency.—When there is dilatation of the stomach due to obstruction at the pylorus, the motor power of the stomach is not sufficient to expel the stomach contents within normal time limits. This is spoken of as insufficiency.

This should be considered a relative insufficiency, as in this type the musculature of the stomach is hypertrophied, especially at the pyloric end, and the contractile power is often increased, but not sufficiently to expel the contents past the obstruction within the normal time.

In the dilated stomach without pyloric obstruction we have the true atony of the musculature, with varying degrees of motor insufficiency.

Some claim that stenosis exists in all cases of dilatation of the stomach, but it is easy to demonstrate that there are two distinct types of chronic dilatation of the stomach differing in symptoms and pathologic findings:

(1) The atonic type of chronic dilatation of the stomach. (2) The stenotic (obstructive) type of chronic dilatation of the stomach.

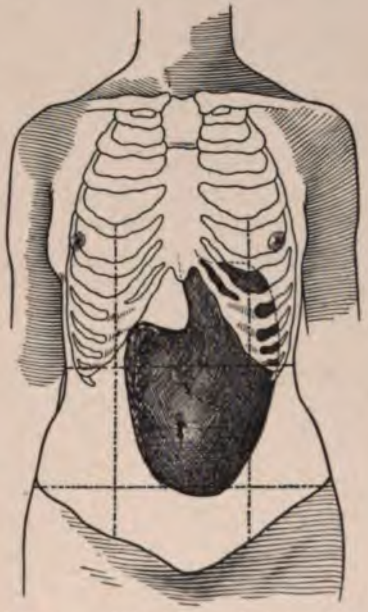

Fig. 149.—Dilatation of the stomach.

Differential Diagnosis.—*Atonic Type.*—In the atonic type there may be few or no symptoms pointing directly to the stomach, the patient frequently suffering from nervous symptoms due to autointoxication and from intestinal disturbances. I have seen many such cases at the Manhattan State Hospital continue a year, or even four or five years, without vomiting. There are no peristaltic waves and cramp-like pains such as occur in the stenotic type followed by vomiting; though some may have dyspeptic symptoms and rarely an attack of vomiting. The post mortem shows the stomach often enormously dilated, with thin walls and no evidence of pyloric stenosis.

Stenotic Type.—In the stenotic (obstructive) type of dilatation dyspeptic symptoms are marked, there are peristaltic waves and cramp-like pains preceding vomiting of large quantities of gastric contents, thirst, etc.

In the benign type of long duration the patient often suffer markedly in nutrition, but the cachexia and other symptoms o malignancy are absent and the disease runs a long course. The post mortem shows considerable hypertrophy of the musculature at the pyloric end of the stomach, and elsewhere thinning and dilatation of the muscular wall with the evidence of stenosis at the pylorus, or constriction from some external factor.

ATONIC DILATATION OF THE STOMACH

Etiology.—As causes, we may have a primary reduction or loss of muscle power or impairment of it from overwork, for example from the ingestion of too much material. It directly follows chronic atony of the stomach, which is the preliminary stage, and hence there are similar etiologic factors, such as wasting disease, tuberculosis, chronic gastritis, heart disease, etc.

Among other causes are bolting the food, frequent overloading the stomach, excessive drinking of large quantities of fluid, especially of those containing much gas; it may rarely be congenital; it is quite frequently associated with rickets, in which case gastroptosis is quite often also present. It sometimes follows repeated attacks of acute atony or acute dilatation.

Nothnagel traces back some cases of chronic dilatation to improper methods of feeding during early life. Atonic ectasy we frequently find among the insane. I have found an enormous number of patients at the Manhattan State Hospital suffering from this condition, and quite a number of the women with gastroptosis, and frequently dilatation associated with it. Examinations of many hundreds of cases during the last five years show that very few stomachs were in the normal position or possessed normal functions. Many of these cases have never vomited and in very many no special symptoms directed attention to the stomach. Unquestionably the *habit of bolting the food common to such patients is a frequent cause of ectasy.* Among the acute melancholics in whom some ultimate cures resulted, auto-intoxication, *in some cases the result of ectasy with fermentation or putrefaction,* was the primary factor in the production of the nervous symptoms. In the epileptic ward one case suffering from dilatation (atonic) with gastroptosis, and absence of free hydrochloric acid, had suffered from numerous epileptic convulsions both day and night, averaging 140 seizures per month. Under simple diet, initial lavage, and at present diet and medication directed to the gastro-intestinal tract alone, she has had no convulsions for two years and a half, with the exception of one week, some fourteen months ago, during which period she was taken off diet and medication while in the general hospital ward with an acute nephritis. Bromids were only given two months at the commencement in small dosage to break the convulsive habit. I have also another case of epilepsy with atonic ectasia and hypochlorhydria, who has gone over a year without a seizure under gastro-intestinal treatment alone.

Even in the incurable insane this atonic type of dilatation has a direct bearing on some of the symptoms. In a series of 13 paretics, examined for the late Dr. Dent at the Manhattan State Hospital,[1] I found 11 cases of atonic dilatation of the stomach and 2 cases of gastroptosis, and in all secretory derangements of the functions of the stomach; 11 of these cases had at some time of the day a temperature of 99.5° F. and upward, in one 102.5° F. Under treatment directed to the gastro-intestinal tract the temperature was lowered in all 11 (in some to normal), the convulsions, which were present in 5 cases, were diminished in frequency, and in one patient suffering from attacks of syncope, cessation of attacks followed treatment.

The atonic type of ectasy is quite common in many nervous cases and is undoubtedly, in some, the cause of the auto-intoxication, and the nervous condition is secondary. Atonic ectasy may, therefore, be in many cases the cause of various nervous conditions. On the other hand, ectasia resulting from insufficient mastication and bolting of food among many of the nervous and insane may be a factor in the production of a vicious circle. Beer drinkers and diabetics suffer from this type of dilatation. Professional men, bankers, and brokers, from their irregular habits and rapid eating, are quite liable to this form. The musculature of the pylorus, as we know, is much thicker than other parts of the stomach wall, and the latter naturally gives way and distends more readily. This does not constitute, however, a stenosis at the pylorus. Ultimately, however, the pylorus itself relaxes in these atonic cases, which undoubtedly accounts for the usual absence of vomiting in these patients.

This type of ectasy is extremely common.

Symptoms are at times not referred to the stomach at all, and often point to the nervous system, or cause an exacerbation of a preëxisting nervous condition. The patient is often neurasthenic or melancholic with the symptoms associated with these conditions. I have seen the lower border of the stomach in atonic ectasy reach nearly to the symphysis. The following symptoms are generally associated:

Constipation usually marked, rarely diarrhea, coated tongue, frequently headache, and at times dyspeptic disturbances, such as belching and pressure after eating, though often these symptoms are absent.

In a few cases of extreme dilatation there may be occasional vomiting of large quantities of fluid. There are no spasmodic pains, no peristaltic waves, and no marked vomiting, as in the stenotic type. In atonic ectasia with chronic gastritis, the gastric symptoms of the latter may be present, in addition to the other symptom already noted. More rarely there may be hyperchlorhydria[1] wit atonic ectasy. These cases suffer from the symptoms of hyperchlorhydria, motor insufficiency is present, there may be spasmodic pain and vomiting of considerable severity, the vomitus being very acid These cases, as already noted, have been mistaken for gastrosuc corrhea. After lavage, and later by aspiration of the contents of th empty stomach, one readily demonstrates that hypersecretion is no present. The symptoms in this type are due to the extreme hyperchlorhydria and to *spasm of the pylorus resulting*. Excellen results are secured by lavage and by the treatment of the hyperchlorhydria. Most of the so-called cures of stenotic ectasia belong I believe, to this class. The mild types belong to the atonic class though they might be considered border-line cases. In the milde cases in which cure results without operation, organic changes at th pylorus, I believe, must necessarily have been slight, while the sever cases with peristaltic unrest and progressive symptoms should b classed under stenosis.[2]

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[1] Proceedings of the American Psychological Association, Sixty-first Annual Meeting, April, 1905; also The Medical News, July 8, 1905.

Gastric Contents.—The gastric findings are variable; fermentation is quite frequently present and hypochlorhydria or nearly complete absence of free HCl; chronic gastritis, occasionally hyperacidity mould has been found, and in cases with gastroptosis and dilatatio at Ward's Island, I have even noted achylia.

The usual manifestations are toxemic in character, with intestina fermentation or putrefaction and indicanuria.

Course.—The milder cases of atonic dilatation are quite amenabl to treatment and can be cured. The severe cases do not run s favorable a course, and I have seen such among the nervous an insane where the dilatation extended nearly to the symphysis. believe that in this type drainage by gastro-enterostomy is indicated since lavage and diet are only palliative.

In the cases of ectasia due to benign stenosis there is at times under treatment, a temporary improvement. The stomach, from a increased hypertrophy of the muscles and a subsidence of the hyperemia at the pylorus, may secure a certain amount of compensatio and empty its contents fairly well for a time, but usually the symptoms return and, finally, resection of the pylorus or gastro-enterostom become imperative in order to save the life of the patient. I hav known these cases to drag along twelve to fifteen years withou operation. They become chronic invalids. Only by operativ procedure can a practical cure of the case be accomplished.

If the stenosis is malignant, then the course depends on th extent of the disease. Even in those cases where removal canno be undertaken, gastro-enterostomy will relieve the symptoms.

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[1] I have found a number of such cases among the epileptics, there being n vomiting or rarely so, and no peristaltic unrest.

[2] These last cases, which do not rapidly respond to treatment of the hyperchlorhydria, should be referred to the surgeon.

OBSTRUCTIVE TYPE (STENOTIC) OF ECTASIA

The stenosis causing this type of dilatation of the stomach may be in the gastric tissue at the pylorus or near the pylorus in the duodenum, or it may be external to the stomach—intrinsic and extrinsic causes. The factor causing constriction may be benign or malignant in character, which would modify the clinical symptoms.

Etiology.—(1) *Congenital stenosis of the pylorus.*

(2) *Acquired stenosis*, such as from ulcer, cicatrices following burns from acids or alkalis; from severe gastritis, causing hypertrophy at the pylorus; repeated spasmodic closure of the pylorus due to hyperacidity (spastic stenosis due to inflammation or irritation); benign tumors; pedunculated polypi; adhesions at the pylorus; external tumors; pressure from large gall-stones (in the gall-bladder); perigastric adhesions; spider-web adhesions (Morris) from the gall-bladder; stenotic hypertrophic gastritis (Boas), a fibrous disease of the pylorus (linitis plastica); sclerosis in the pyloric end of stomach (Ottinger); pressure from external tumor; malignant disease of the pylorus.

(3) *Stenosis of the duodenum* from ulcers, cicatrices, carcinoma, external compressions, adhesions, kinks, or diverticula.

It is the modern consensus of opinion that bile may regurgitate into the stomach even if the stenosis is present at the pylorus, as the cicatricial tissue holds it open in many cases; so its presence or absence does not always aid us as to locating whether the stenosis be pyloric or duodenal. Continuous regurgitation of bile and pancreatic juice, however, is suggestive of obstruction of the duodenum below the common duct.

Movable kidney I do not believe has any relation to ectasy, but when associated with so-called dilatation, it can be demonstrated that the latter is really a gastroptosis.

Pathology.—In the stenotic type, the musculature at the pyloric end of the stomach is much thickened; the fundus is much thinner than normal. The pathologic findings at the pylorus vary according to the cause of the stenosis. The intestines are pushed downward and the liver slightly upward when marked ectasy is present.

Symptoms.—The symptoms of dilatation of the stomach due to pyloric stenosis are quite characteristic, but are modified if the condition is cancerous, or in the special type described by R. T. Morris.

The usual symptoms are thirst, dryness of the throat, dry skin, oppression, feeling of cramp-like pains of considerable severity, generally associated with peristaltic restlessness of the stomach, eructation of odorous gas, vomiting of considerable chyme, often containing remnants of food taken the day before. This may occur from once to several times a day. The bowels are extremely constipated. Emaciation may become very marked and the loss of adipose on the abdomen so great that the skin is in dry wrinkled folds. The urine becomes markedly diminished in advanced cases its reaction is frequently alkaline.

Intestinal fermentation and putrefaction with indicanuria are often present.

Bradycardia and dyspnea (cardiac asthma) at times are present as are also stupor, headache, and so-called gastric vertigo.

Tetany, or epileptiform attacks may complicate the condition.

The benign type of stenotic dilatation is characterized by rather a long course, with often considerable temporary improvement *under treatment, with a tendency to relapse.*

In the *malignant type* we have the marked cachexia and rapid loss of weight within a few months, the age of patient usually over forty or forty-five, and the character of the vomitus to be noted later.

In my experience the cases characterized by attacks of gastrosuccorrhea are more frequently associated with ulcer at the pylorus with benign stenosis.

Special Type.—In an interesting type, to which Robert T. Morris has called attention, there may be vomiting of considerable blood, pain, etc., suggestive of an *active ulcer.* Gall-bladder spider adhesions have been found to be the factor. The symptoms are probably explainable by circulatory interference. The history of previous gall-bladder disease should be looked into.

Examination of the Gastric Contents in Malignant Pyloric Stenosis.—Coffee-ground vomit, or the presence of occult blood; free HCl markedly diminished or absent, lactic acid present; few or *no sarcinæ* and the Boas-Oppler bacilli present, in connection with the clinical symptoms, are diagnostic of malignant stenosis. Meat is undigested. Free HCl may be present, especially in the early stages, or when the carcinoma is engrafted on an ulcer.

With benign stenosis we have: the gastric contents separating into three layers, the upper being gaseous; HCl is marked (hyperacidity); yeast and *sarcinæ* are abundant; undigested starch is present. Mould may be found and occasionally bile or sulphuretted hydrogen.

I agree with Einhorn that bile can enter the stomach in some cases of stenosis of the pylorus, the thickened tissue allowing a slight patency.

DIAGNOSIS

Ectasy means dilatation of the stomach combined with motor insufficiency.

We must, therefore, first determine the position of the organ. Frequent errors have been made in differentiating dilatation and gastroptosis. The position of the upper border of the stomach is the chief point.

There is a simpler method which is dependent on whether or not a movable kidney be present. The movable kidney is almost invariably part of a general ptosis of the viscera (splanchnoptosis), and movable kidney from traumatism is extremely rare.

If we find a movable kidney with a stomach whose lower border is in an abnormal position (too low down), the diagnosis is gastroptosis. If no *movable* kidney is present and the greater curvature is low down, plus motor insufficiency and symptoms, the condition is one of dilatation.

An extremely simple method of locating the lower border of the stomach is by the *splashing sound* (succussion). This has been fully described, and also the creation of the splash for diagnosis. Dehio's method can be used as a check.

We may employ in addition:

(1) *Inspection*, which will in some cases show the outline of the distended stomach, especially after distention with carbonic acid gas. *Active peristalsis is also evident on inspection and is diagnostic of pyloric obstruction.*

(2) *Palpation.*—By this means the peristaltic movements may at times be felt, as can also the cushion-like resistance of the distended stomach. Occasionally a small oval tumor can be determined in benign stenosis, though generally it is not appreciable. In carcinoma of the pylorus the hard resistant mass can often be appreciated.

(3) *Percussion* and *auscultatory percussion*, especially before and after the addition of water, as already described, are useful. The "scratch method" is of service.

(4) *Inflation.*—The stomach may be inflated with air or carbonic acid gas. The outlines can thus be more readily determined and the position of the upper curvature mapped out. Inflation with air or water renders a tumor, if present, more evident to percussion and palpation if it lie on the anterior wall. It disappears if posterior.

(5) *Transillumination.*—This method, especially with the circumscribing gastrodiaphane and fluorescein, readily determines the outline of the stomach and differentiates between dilatation and gastroptosis in disputed cases.[1] The use of *mensuration by means of stiff sounds is deprecated.*

The determination of the motor functions is most important, *as motor insufficiency is a salient feature.* There are different degrees of this motor insufficiency, and this is best determined by the test breakfast or test meal.

Test Breakfast.—Ewald's test breakfast. Aspirate the contents one hour later.

(1) *Normal position of the stomach*, with a residuum aspirated of 100 cc. or over, and symptoms, show atony of the stomach.

(2) Descent of the lower border of the stomach; 100 to 150 to 200 cc. residuum or more with symptoms and with kidneys in normal position show dilatation of the stomach.

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[1] The *x*-rays give no additional information over the methods described, and the expense is an objection.

(3) If movable kidney, gastroptosis is present.

Six or seven hours after Leube's test meal the healthy stomac should be found empty. If undigested food is found (200 to 600 cc or more), insufficiency is present and the degree of insufficiency i indicated by the amount of residuum.

It is always preferable to wash the stomach before the test meal so as to get rid of the old residuum and make an accurate test. I considerable residuum be found at the end of seven hours, a furthe test should be made. Wash the stomach and directly thereafter giv at 10 P. M. a light supper—a little soup, a slice of bread, a slice o beef, and a little chopped spinach; a small amount of boiled rice o a dozen raisins may be substituted for the spinach. Aspirate an wash the stomach twelve hours later before breakfast. In som cases there will be a marked residuum after seven hours, *but non after twelve hours;* in others there will also be considerable afte twelve hours, showing different degrees of insufficiency.

The stomach should be washed, as well as aspirated, to remov all the contents.

The salol and olive oil tests are not as reliable.

TABLE OF DIFFERENTIAL DIAGNOSIS

	Atonic Dilatation.	*Stenotic Dilatation.*	
		Benign stenosis of pylorus.	*Malignant stenosis of pylorus.*
Duration.	Long unless recently acquired.	Quite long, two years, generally considerably more.	Short, few months to one year or one year and a half.
Course.	Long, often.	Generally intervals of quiescence or improvement.	Progressive.
Tumor.	None.	Occasional (small and smooth).	Present; later palpable.
Pain.	Absent.	Spasmodic attacks.	Always present and exacerbations.
Peristaltic restlessness.	None.	Marked.	Present and at times marked.
Vomiting.	Most frequently absent.	Frequent.	Fairly frequent.
Cachexia.	None, but some loss of weight.	None, but great emaciation.	Present.
Symptoms.	Often toxemic, referred to nervous system and not specially referred to stomach; at times gastric.	Marked gastric.	Marked.
Blood.	None in vomitus.	None except in gall-bladder adhesion cases.	Coffee-grounds in vomit or occult blood.[1]
Gastrosuccorrhea.	Rare.	More frequent with ulcer.	Generally absent, though occasional.

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[1] Blood or occult blood present in stool in cancer.

GASTRIC CONTENTS

	Atonic Dilatation.	*Benign Stenosis.*	*Malignant Stenosis.*
Total acidity.	Lessened; more rarely increased.	Increased.	Generally diminished.
Free hydrochloric acid.	Often hypochlorhydria; more rarely hyperchlorhydria.	Generally increased.	Usually absent.
Lactic acid.	At times present.	Absent.	Present, marked usually.
Fermentation.	Often marked.	At times marked.	At times, depending on location of growth.
Odor.	Often present.	Unpleasant.	Fetid at times.
Boas-Oppler bacilli.	Occasional.	Rare.	Usually present.
Mucus.	At times, if gastritis.	At times, if gastritis.	In some cases.
Sarcinæ.	Present often.	Present markedly.	Usually absent.
Yeast.	Marked often.	Often present.	Pronounced yeast fermentation rare.

TREATMENT

The treatment of chronic dilatation of the stomach varies considerably, depending on whether it be due to atony or to benign or malignant stenosis.

Atonic Dilatation.—This is by far the most frequent type of dilatation which we are called upon to treat, especially among bankers, brokers, and professional men, who habitually overeat, bolt their food, or are heavy drinkers. Associated with or having a direct bearing on this condition, we may find hypochlorhydria, hyperchlorhydria, or, at times, chronic gastritis. Some of these cases are, in their incipiency, of rather mild type, and prophylaxis, as regards avoiding rapidity of eating and eliminating indigestible food and overeating, is of value. If the patient is run down or anemic, iron and tonic treatment are indicated.

Diet.—Though some have recommended a so-called dry diet in dilatation of the stomach, it is a well-known fact that liquids are first evacuated from the stomach, then mushy food, and finally solid food, and this scientific knowledge should be our guide in feeding such cases. Water and food soluble in water leave the stomach soonest of all.

Large quantities of fluid should not be given at a time lest they overdistend the flaccid stomach, but if they are administered in smaller quantities at frequent intervals, a considerable amount can be employed.

It has been demonstrated that alcohol, sugar, and dextrin cause a secretion of water into the stomach.

Milk has been usually recommended as the standard diet in this condition as possessing highly nutritive properties, and the statement has been made that it does not stay in the stomach much longer than plain water.

Penzoldt has demonstrated that water, cocoa, meat broth, soft-boiled eggs, and boiled milk (100 to 200 gm.) leave the healthy stomach within one to two hours, cooking altering the curd formation.

Raw milk takes a considerably longer period, and curds hav been found frequently in the normal stomach two or three hour after ingestion.

Experiments have been conducted on my service at the Manhatta State Hospital in cases of dilatation of the stomach, and the period for the raw milk to remain in the stomach were investigated. Afte three hours large masses of curd were aspirated.

If the milk were diluted one-half with water, the residuum foun at a certain period was just one-half as much as when pure raw mil was used, which formed curds. The higher the dilution, the greate the quantity passed from the dilated stomach within a definite time Another objection is that 1 liter of milk only represents about 64 calories, and too large an amount would be required if sufficien nutrition is to be obtained from milk alone. Strained soups an strained gruels are evacuated more rapidly. If milk be given, i should, preferably, be combined with some strained gruel or th latter made with milk, so that the nutritive value may be increased.

In the severer type of cases the diet suggested by Seibert i typhoid appeals strongly to the author. It possesses considerabl nutritive value, namely:

Strained rice, ℥viij (250 cc.), barley or oatmeal soup containin the extract of ½ pound of meat and the yolk of a fresh egg. Thi can be spiced slightly to improve the flavor. It can be given fiv or six times daily.

Strained pea soup, lentil, tomato, or potato soup can be used in addition.

Rice flour is excellent in the form of a thin gruel, and can be made with milk which has been thoroughly boiled. The object should be to give frequent (five or six smaller) meals, so as not to overburden the stomach, and yet secure a sufficient amount of nutrition to improve the patient's physical condition.

Cream, ℥ij (60.0) in ℥iv (125 cc.) of water, possesses considerable nutritive value.

Crackers heated thoroughly and well buttered can be rubbed up in the broth.

Fat in the form of cream and butter should be administered.

In the *milder cases*, scraped beef, rare beef, soft-boiled eggs thickened with *a small amount of mashed potatoes*, and rice strained through a colander, with plenty of butter, can be given, with a little asparagus and spinach. Other vegetables are more difficult to expel from the stomach.

Matzoon, kumyss, bacillac, kefir, and milk prepared with lactone tablets (lactone-buttermilk) are of special value in cases suffering from auto-intoxication having nervous symptoms and indicanuria (intestinal putrefaction). The matzoon can be diluted with one-third water or Vichy that has been allowed to become flat. This last avoids gaseous distention of the atonic stomach. It is also preferable to allow some of the gas to pass off from the kumyss. About 1 quart of one of these preparations can be used daily—additional water or Vichy one-third in volume being then added. These sour milk preparations, especially with the slight dilution, pass readily from the stomach. They do not curdle like plain milk. The yolks of several raw eggs, stale bread or crackers with plenty of butter and cream, strained vegetable soups, rice gruel, and sanatogen can be added. Meat preparations should be avoided in these cases.

In cases with deficiency of hydrochloric acid, the meats are not well digested and should be given in smaller quantities; rice, barley, and tapioca (strained) or in purées, and mashed potatoes are of service, and in larger amounts. I have often found raw eggs beaten up in water or milk of great service, employing at times six to eight daily. The milk can be completely or partially peptonized to lessen curd formation.[1]

If thirst is marked, rectal enemata of hot normal saline solution are indicated,[2] and in very severe cases the stomach may be given a rest and nutritive enemata be given for a few days.

Sanatogen, preferably flavored, tropon, and somatose are useful adjuncts, given in divided doses in the broths. If anemia is present, iron tropon can be added, ʒj (4.0), three times a day. Always peptonize milk if given by enema.

After eating the patient should lie down for from one-half an hour to an hour, preferably on the right side, so the stomach can empty itself more readily.

Mechanic Support.—One of the most important methods of treatment is the use of proper support to the organ, and the ideal method is by Rose's adhesive plaster belt, a description of which has been given. It increases intra-abdominal pressure and the stomach is pushed upward, acting in effect like gastroplication. Transillumination was employed in one case (Fig. 150), the belt applied, and transillumination again carried out (Fig. 151).

The illustration (Fig. 151) shows the result, the lower border of the stomach being elevated 4 inches. This belt should be worn four or five weeks and a new one then applied. It aids in the evacuation of the stomach contents. Silk abdominal belts may be substituted, but the support is not continuous and the adhesive strapping is superior.

General Hydrotherapy.—External douches—the fan douche and also the Scotch douche—applied to the region[3] of the stomach and changing the temperature of the water has been serviceable in some cases. Cold compresses and cold sponging are at tim useful.

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[1] With the precaution noted, milk may be employed in the cases with not too marked motor insufficiency. It is a simple matter to test whether it leaves the stomach readily or not.

[2] Proctoclysis is of value.

[3] An adjustable silk belt is worn in this event.

Local Treatment of the Stomach.—(1) *Lavage.*—In some of t milder atonic cases proper diet, mechanical support, and appropria medication may suffice without or with occasional lavage. In t more severe cases lavage is indicated, and the time of its performan and frequency depend upon the degree of dilatation and the amou of residuum found after the test meal (degree of motor insufficiency

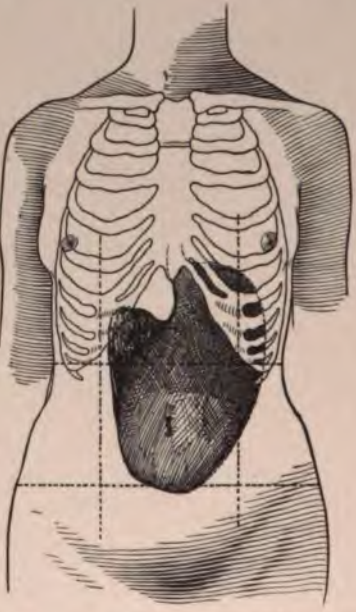

Fig. 150.—Dilatation of the stomach. Transillumination with fluorescein before application of Rose's belt (Case 1) (Ward's Island Gastro-intestinal Clinic, Manhattan State Hospital).

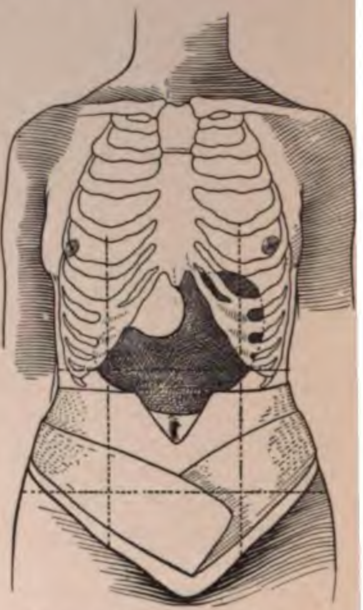

Fig. 151.—Dilatation of the stom ach, same patient (Case 1). Trans illumination with fluorescein afte application of Rose's belt. By accu rate measurement the stomach ha been elevated and the lower border i 4 inches higher than it was before th belt was applied. The lower borde now lies above the umbilicus (Ward' Island Gastro-intestinal Clinic, Man hattan State Hospital).

If there is not immediate improvement if lavage is omitted, it must be added to the treatment.

If a large amount of residuum is present after a test meal or test breakfast, or there are nervou symptoms, or in the morning before breakfast a residuum is present lavage should be carried out.

As to the proper hour for lavage, I believe Riegel holds soun views, and my own experience agrees with his; if the residuum i 200 to 500 cc. or more before supper, it is best to wash the stomac then and follow with a light meal; if this is not done the organ wil contain fermenting food during the night which will increase th atony. If food is present before breakfast a second lavage is then indicated.

The washing should be performed with the patient both sitting and lying down, so as to remove all the irritating material. The stomach should be washed until the return is clear.

Daily lavage is generally necessary at first. As the tone of the stomach improves, the residuum found will decrease and washing may be performed less often. The first part of the washing may be done with plain warm water, though normal saline solution is preferable.

I have found milk of magnesia (Phillips), ℥ij (60.0) to 1 quart (liter) of water, excellent for the preliminary lavage. It is well to employ antifermentatives in the final treatment, such as—

Acid salicylic	1: 1000
Sodium salicylate	1: 1000
Sodium benzoate	1: 1000
Listerin	ʒj (4.0) to 1 quart (liter).
Glycothymolin	
Borolyptol	
Resorcin or	gr. 15 to 30 (1.0–2.0) to 1 quart (liter).
Boric acid	

(2) *Electricity.*—The intragastric faradic current (preferably) can be employed if there be no objection on the part of the patient, using Lockwood's instrument. In many cases the percutaneous method is advisable.

Static electricity is claimed to be of value, and in some cases to reduce the size of the atonic dilated stomach.

Massage or *vibratory massage* are of service to tone the musculature and aid in emptying the stomach.

(3) The *stomach douche* has been recommended in the milder forms to stimulate the organ. The fluid should be at a temperature of 95° to 85° F. (gradually reduced).

If hydrochloric acid is diminished, normal salt solution may be employed; if HCl is increased, then use silver nitrate 1 : 3000 to 1 : 2000.

Bitter remedies, such as quassia (fluidextract), 0.888 cc. (♏15) to 1.77 cc. (ʒss), or a cup of quassia-water (quassia cup filled with water and allowed to set for half an hour); or hops (fluidextract lupulin), 0.888 (♏xv) to 1.77 cc. (ʒss), or fluidextract of condurango, 1.77 cc. (ʒss) to 3.54 cc. (ʒj) to a liter of water, have been recommended for lavage as a stimulant, but I see no special value in their use.

The *stomach spray* has also been suggested in place of the stomach douche, but it possesses the disadvantage of injecting considerable air.

Medicines.—If there is deficiency of hydrochloric acid, the stomachics and hydrochloric acid should be administered, such as are employed in chronic gastritis. The following prescription is often valuable:

℞. Tinct. nucis vomicæ }
Acid hydrochloric, dilute } .... āā 12.0 (ʒiij)
Comp. tinct. cinchona........................ 16.0 (℥ss)
Aq. destil.......................... q. s. ad. 125.0 (℥iv).—M.

Sig.—One to two teaspoonfuls in a wineglassful of water t. i. d. half an ho
before meals.

If there is hyperacidity, magnesia usta, gr. xv to ʒss (1.0–2.0)
more, in water t. i. d. an hour after meals. Or milk of magnes
(Phillips), ʒj to ij (4.0–8.0), in water. These are excellent remedie

If bicarbonate of soda be employed, it is better to combine
with magnesia usta, equal parts, as it readily generates carboni
acid gas.

In the *atonic type* of dilatation I have always been inclined t
employ nux vomica or its alkaloid as a stimulant to the musculatu
of the stomach, even though hyperacidity be present, which is rar
in the latter event combining belladonna with the nux, as a pil
before meals:

℞. Ext. nucis vomicæ }
Ext. belladonna } .... āā gr. ¼ (0.016).—M.

or

℞. Strychnin.............................. gr. 1/60 (0.00108)
Atropin................................ gr. 1/100 (0.00064).—M.

If nux vomica is incorporated in the stomachic mixture, it shoul
not be repeated. If the tincture of nux vomica is employed, it ca
be gradually increased to large dose—ʒss (2.0) t. i. d.

For *fermentation* and intestinal putrefaction the following remedie
are of service, given three times a day half an hour to an hour afte
meals: Salicylate of soda, benzoate of soda, ichthoform, ichthalbin
bismuth salicylate, salol, resorcin, benzonaphtol, bismuth phenolate,
or bismuth sulphocarbolate, all in doses of gr. v (0.3) each. Urotropin,
gr. v (0.3), given in combination with sodium benzoate, gr. v (0.3), in
water t. i. d. after meals is also useful.

I have found resorcin an excellent remedy, alone or combined with bismuth subnitrate. If mould is present the creosote preparations as suggested by A. Rose are preferable, such as beechwood creosote, ♏j (0.059 cc.), or carbonate of creosote (creosotal), gr. 5 (0.3), three times a day after meals.

For *constipation* the olive oil injections at night, to be retained, ℥iv (125 cc.) to 1 pint (500 cc.) or more, as suggested by Fleiner; massage, electricity, the establishment of a regular hour for stool, the administration of a glass of water on rising, and, if required, the use of the cascara preparations or the aloin and belladonna pill, or one of the phenolphthalein preparations, such as phenolax, at night. The saline cathartics are objectionable.

Gastrosuccorrhea is rare in the atonic type of dilatation. In the event of its presence, lavage with nitrate of silver (1 : 2000) twice a week and belladonna tincture, ♏x (0.592 cc.) t. i. d., or extract of belladonna, gr. ⅓ (0.022) t. i. d., are indicated. For further treatment, the chapter on this subject should be consulted.

For gastric tetany, which may occur in the atonic type of ectasy, but which is a rare condition, lavage is of service, but gastro-enterostomy is indicated. Moynihan has operated on 14 cases of gastric tetany, with cures in all.

Surgery.—In atonic ectasy, when no improvement occurs under treatment or when the dilatation is of great degree and the patient's condition seems to be getting worse, operation is indicated—preferably drainage of the stomach by gastro-enterostomy.

Gastroplication—infolding the wall of the stomach and sewing it in pleats—has also been successfully reported. Coffey has sutured the greater omentum to the abdominal wall and thus supported the stomach in a hammock. He reports two favorable results.

Treatment of Stenotic Dilatation (Benign Stenosis)

In these cases, where there is a mechanical obstruction to the exit of the gastric contents, the muscular action is increased during the earlier stages and hypertrophy of the pyloric end of the stomach is present; later the fundus and body become distended and thinner. Gentle massage from left to right, or vibratory massage by the same method, the patient lying on the right side and the manipulations being performed an hour or two after meals, may aid in emptying of the stomach. Electricity is of slight or no value. I have seen no permanent benefit from these methods in the obstructive type of dilatation. Olive oil, ℥ij to iv (60.0–125 cc.), administered three or four times a day before meals will aid the passage of the food through the stenosed region, and Rose's adhesive plaster belt is also of service.

The diet must be liquid in the worst cases and mushes may be employed in the less severe types. Raw eggs, six to eight a day, plenty of fat, such as butter and cream, sanatogen, somatose, and tropon, are all of service. The general method of feeding with small frequent meals is the same as in atonic ectasy. Improvement in weight must be secured. Solid food is objectionable. "The sour milks, such as bacillac, lactone-buttermilk, kefir, matzoon, and kumyss, administered after the manner described in atonic ectasy, are of value." Lavage is always necessary, and for the attacks of spasmodic pain is the most rapid method to secure relief. As hyperacidity is present, alkalis are indicated to correct this condition, as in hyperchlorhydria.

Tincture of belladonna in large doses, 10 to 15 drops t. i. d., will often relieve spasm and pain, also the application of heat.

Vomiting.—For vomiting lavage is indicated, followed for several days by rectal feeding and then commencing with a small amount of liquid nourishment.

Rectal injections[1] of normal saline solution, ℥viij to Oj (250–500 cc.), may be indicated to relieve thirst and collapse.

Thiosinamin, gr. v (0.3) doses t. i. d. by mouth, or gr. iij (0.2) doses in 15 per cent. alcoholic or 10 per cent. glycerinated solution by hypodermic, may prove of service in fibrous contractures of the pylorus.

Gastrosuccorrhea may occur in cases when there is an ulcer with stenosis at the pylorus and, rarely, hemorrhages. Temporary treatment, as for hemorrhage of gastric ulcer is indicated, and then surgical procedure. The treatment for gastrosuccorrhea is described in the chapter on that subject.

Gastric tetany may be a complication for which lavage is indicated temporarily, and then operation (gastro-enterostomy).

I have seen cases of ectasy from benign stenosis lose 75 pounds in weight and regain 50 to 60 pounds under treatment, and ultimately relapse and come to operation. I have treated many so-called brilliant cures by specialists, the ultimate results being the same. These patients, even at the best, tend to become chronic invalids and always require treatment. The best physician for these cases is the *surgeon*.

If the cause of the stenosis lies external to the pylorus, bands, adhesions, etc., can be separated. If it is intrinsic, resection of the pylorus can be performed in suitable cases, or drainage by gastro-enterostomy. The latter is usually the operation of selection. Divulsion I do not approve.

I have seen a patient gain 100 pounds in weight in eight weeks after gastro-enterostomy, and, from being a confirmed invalid, restored to perfect health.

MALIGNANT STENOSIS

In these cases early radical operation is indicated, as described under Cancer of the Stomach; otherwise, palliative gastro-enterostomy. If operative procedure be refused, then diet, lavage, and the treatment laid down under Carcinoma Ventriculi.

COMPLICATIONS OF CHRONIC ECTASY

GASTRIC TETANY

Tetany is characterized by peculiar bilateral tonic spasm of the extremities, either paroxysmal or continued.

Pathology.—In all cases there is dilatation of the stomach of a high degree, due generally to stenosis of the pylorus or the duodenum; frequently the result of an ulcer; rarely from carcinoma.

Gastrosuccorrhea has been associated with it in some cases. Tetany has also been reported with acute ectasy and in atonic ectasia.

Etiology.—There are three theories as to its cause:

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[1] Proctoclysis is of service.

(1) Kussmaul and, later, Fleiner believed its symptoms are due to the great loss of fluid in the system, the thickening of the blood, and the consequent drying of the tissues.

(2) Friederick Müller and Germain Sée consider it to be the result of some reflex action, as Müller brought on an attack by tapping the epigastric region. Riegel has observed it on passing the stomach-tube; and it has also occurred in cases of intestinal worms.

(3) The third theory explains it on the ground of auto-intoxication, since fermentation and putrefaction are present in the stomach.

This last is probably correct, as the cases have been benefited by lavage and cured by stomach drainage (gastro-enterostomy).

Amato reports a case of gastric tetany with death. He has introduced fermenting materials into the stomachs of animals and produced dyspnea, myosis, muscular contraction, and trismus. The liver and pancreas (post mortem) showed lesions, such as are usually found in poisonings and intoxications.

Symptoms.—There are tonic and clonic bilateral spasms, which appear suddenly and are generally confined to the extremities, the flexor muscles being chiefly affected. The fingers are bent at the metacarpophalangeal joint, extended at the terminal joints, being pressed close together, and the thumb is contracted into the palm of the hand. The wrists are flexed, the elbows bent, and frequently the arms are folded over the chest. The knees are bent, the feet extended, and the toes adducted.

In severe cases there may be trismus and the angles of the mouth are drawn up. There is sometimes edema of the hands and feet.

The spasms are usually paroxysmal and last for a variable time. The eyes may be turned up.

In the acute attack there may be a rise of temperature and elevation of the pulse. In some cases there may be involvement of the muscles of the back and of the thorax, with dyspnea and cyanosis.

The attack may be acute, from a few minutes to several hours, but there may be some stiffness and contraction lasting several weeks.

Diagnosis.—There are certain diagnostic features:

(1) "*Trousseau's Symptom.*"—As long as the attack is not over, the paroxysms may be produced by compressing the affected parts, either in the direction of their principal nerve-trunk or over their blood-vessels, so as to impede the venous or arterial circulation.

(2) "*Chvostek's Symptom.*"—There is an increase in the mechanical excitability of the motor nerves. A slight tap over the facial nerve will throw the muscles to which it is distributed into active contraction.

(3) "*Erb's Sign.*"—The electric irritability of the motor nerves, to the galvanic current especially, is increased.

(4) "*Hoffmann's Sign.*"—Heightened excitability of the sensory

nerves. The slightest pressure may cause paresthesia in the regi of distribution.

The **prognosis** of tetany is extremely bad.

Frequency.—Moynihan believes it to be not so very rare, ar reports 14 cases in which gastro-enterostomy was performed, wi a cure in each case; though some claim only 30 to 40 cases are r ported.

Tetany-like attacks with epileptiform attacks are more frequen and will be referred to under Epilepsy.

Treatment.—Bromids, and even chloroform inhalation durin the acute attack. Lavage is beneficial.

The chief indication is drainage of the stomach by gastr enterostomy.

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CONVULSIONS—EPILEPSY

Epileptic seizures occur both in cases of chronic ectasy and i other affections of the stomach. I have a case at present unde observation at the Manhattan State Hospital who suffered fro repeated attacks of epilepsy, 140 seizures a month. There is dil tation of the stomach, with ptosis and hypochlorhydria. For th last two years, while having occasional lavage, dietetic treatmen with medication directed to the stomach, no bromids, she has had n attacks, except for the period of a week, a year and a half ago, whe the treatment was omitted. At the same institution there is anothe patient with atonic ectasia and hypochlorhydria, who had frequen epileptic attacks and who has had no seizures for over a year, as result of treatment directed to the gastro-intestinal tract.

I have reported a case of hyperchlorhydria,[1] with epilepti seizures, apparently cured by appropriate treatment; and cases o dementia paralytica with chronic ectasy, in which the convulsion were diminished under treatment and the high temperatures returne to normal. I have also seen *tetany-like convulsions* in a case o dementia præcox, with gastroptosis and marked ectasy, improv after treatment of the stomach. Salt-free diet, the avoidance of meat, open air, and treatment appropriate to the gastric findings in each case are indicated.

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[1] Observations on the Relation of the Gastro-intestinal Tract to Nervou and Mental Diseases, reported April 17–21, 1905, American Medicopsychologica Association, and in their proceedings.

In conclusion I cannot recommend too highly the sour-milk diet in the treatment of these cases. Matzoon, kumyss, kefir, or the lactone-buttermilk can be employed after the manner indicated under Atonic Ectasy. I have found Wm. H. Thomson's formula for the preparation of matzoon of great value. It is as follows:

"1. Place half a cake of yeast in 1 pint (500 cc.) of fresh milk in a pitcher covered with a towel in a warm place for twelve hours; then

"2. Add to this 1 quart (liter) of milk and keep in a warm place for twelve hours; then

"3. Take 1 pint (500 cc.) of No. 2, add to it 1 quart (liter) of milk and keep in a warm place for twelve hours; then

"4. Take 1 pint (500 cc.) of No. 3, add to it 1 quart (liter) of milk and keep in a warm place for twelve hours.

"This makes 1½ quarts (1500 cc.) of matzoon, the entire process occupying forty-eight hours.

"One quart (liter) of this can be administered in divided doses daily, breaking in it stale bread or crackers and eating it with a spoon. The remaining pint (500 cc.), with the addition of 1 quart (liter) of milk at the end of twelve hours, will furnish 1½ quarts (1500 cc.) of matzoon. Fresh matzoon can be made daily from the former pint (500 cc.) of mother matzoon for about two weeks, when the process must be started over again. If larger quantities are to be used, 1 quart (liter) of No. 2 can be used with 2 quarts (liters) of milk, and so on."

This method has been extensively employed by Thomson and the author in feeding our epileptics. One can also use ℥iv to vj (125–185 cc.) of the ordinary bottled matzoon to 1 quart (liter) of milk, which will produce matzoon if kept twelve hours in a warm place. From this daily matzoon can be made from each previous supply for about a week, when a fresh bottle must be employed.

CHAPTER XVI

ANOMALIES IN THE POSITION AND FORM OF THI STOMACH—HOUR-GLASS STOMACH—DISLOCATIONS—GASTROPTOSIS

ANOMALIES of form are frequently congenital. Fore-stomach i a dilatation of the lower end of the esophagus immediately above th diaphragm. Antrum cardiacum is a sacculated diverticulum o the esophagus situated below the diaphragm. In some cases n symptoms are present; in others food becomes lodged and cause serious results. Megalogastria is a congenital or acquired larg stomach with normal functions. Microgastria is an abnormally smal stomach with normal functions. Angustatio ventriculi is an extremely small stomach due to stricture of the cardia or esophagus o cirrhosis ventriculi.

Congenital Narrowing of the Pylorus.—This condition is du to hypertrophy of the circular muscles of the pylorus. It may b of so severe a type that ingestion of food may be impossible and th infant die within a few days after birth. In milder cases chroni ectasy may result.

HOUR-GLASS STOMACH

This condition may be *congenital*, but is more frequently acquired The stomach has a peculiar sacculated outline, and is divided int two parts—the cardiac and pyloric (Fig 152). In some cases the cardiac sac i larger, in others, the pyloric.

Etiology.—Cicatrized tissue contraction following an ulcer; less frequentl peritonitic adhesions, corrosive gastritis and carcinoma are causes. Slight form may produce no characteristic symptoms In advanced cases the division may be recognized. By the ingestion of bismuth and the use of the fluoroscope this condition can be demonstrated.

The following diagnostic points have been given by Moynihan:

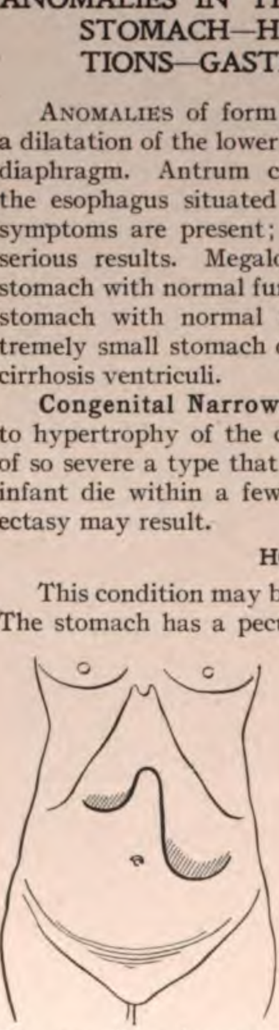

Fig. 152.—Hour-glass stomach.

With lavage, part of the fluid is lost; if the stomach is washed clean, a sudden reappearance of the stomach contents takes place, "paradoxical dilatation"; when the stomach has been apparently emptied, a splashing sound may be elicited by palpation of the pyloric segment; after distending the stomach, a change in the position of the distention tumor may be seen in some cases. Gushing, bubbling, or sizzling sounds are heard on dilatation with carbonic acid gas at a point distinct from the pylorus. In some cases, when both parts are dilated, two tumors with a notch or sulcus between are apparent to sight and touch. On both sides of the furrow there will be a loud tympanitic sound which cannot be elicited in the middle.

Stockton finds that if the first part of the stomach is aspirated, after manipulation, it is sometimes possible to force from the second into the first portion a gastric juice of different quality. Operative procedure is the only method of cure.

DISLOCATION OF THE STOMACH

The fundus may be dislocated upward. Among the causes are:

Absorption of a pleuritic exudate on the left side; after contraction of the lung or any process which is accompanied by upward dislocation of the diaphragm; excessive distention of the abdominal cavity, forcing the diaphragm upward, such as from pregnancy, ascites, tumors, and meteorism; diaphragmatic hernia.

The cardiac end of the esophagus may become bent. Lateral dislocation is rare, and may be caused by tumors of the spleen, distended colic flexure, or lateral pressure from an enlarged liver. As a rule, the latter forces the stomach downward.

Downward dislocation (gastroptosis) is the common form.

GASTROPTOSIS—ENTEROPTOSIS—GLÉNARD'S DISEASE

(*Synonyms.*—Gastroptosia (Rose); Visceroptosis; Splanchnoptosis; Abdominal Relaxation or Atonia Gastrica (Rose); Atony of the Third Degree.)

Definition.—Gastroptosis may be defined as a prolapse or downward displacement of the stomach, right kidney or both kidneys, and other organs of the abdominal cavity,[1] which may be associated with disturbances of the gastro-intestinal tract and pelvic organs, together with various nervous symptoms.

Nephroptosis is a stigma of gastroptosis.

Introduction.—I here use the term "gastroptosis" with the usual definition (ptosis of the stomach), though Rose has shown it correctly means descent of the belly (splanchnoptosis). The reader must remember that gastroptosis is a quite frequent condition, and that it may be accidentally discovered in some cases which have no symptoms whatever. On the other hand, there are various degrees of ptosis of the stomach, in some of which the symptoms are rather mild in character, while in others there may be the symptoms-complex of Glénard's disease.

It is not the position of the lower border of the stomach which constitutes a ptosis, but that of the upper border; with the relaxation of the suspensory ligaments of the stomach the lesser curvatur sinks as well as the greater, and we may have varying degrees o ptosis, from moderate obliquity of the upper border to a vertica stomach; while on the other hand, the entire organ may sink and giv the crescentic form of gastroptosis. The determination of the lowe border alone is not diagnostic, since it may merely be evidence of dilated stomach. Hundreds or even thousands of cases of nephroptosis have been reported with dilated stomachs, the dilatation bein imputed to pressure of the kidney on the duodenum, and no investigation has been made of the position of the lesser curvature.

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[1] Ptosis of the heart may also occur.

From my own experience, I do not hesitate to say that movabl kidney (nephroptosis), with the lower border of the stomach lowe than normal, is diagnostic of gastroptosis. Dilatation of the stomach is often associated with gastroptosis, but kidney pressure on th duodenum, in my opinion, has no bearing as to its production. Furthermore, treatment for gastroptosis will generally cure thi condition. Ptosis of the stomach in some of these cases may be o extremely mild type.

Anatomic Considerations.—It is necessary to briefly allude t certain anatomic features. The liver, as we know, is suspended from the diaphragm by ligaments derived from the peritoneum. The cardiac end of the stomach is held quite fixedly in position by the esophagus, and there is a peritoneal attachment to the diaphragm at this point, the gastrophrenic ligament. In this location the stomach lies in close relation to the diaphragm, while the lesser curvature is suspended from the liver by the lesser omentum (gastrohepatic). The spleen lies in close relation to the diaphragm, being attached thereto by ligaments (processes of the peritoneum), and to the stomach by the gastrosplenic omentum.

It is thus readily understood how compression of the lower part of the thorax or effusions above the diaphragm may mechanically force down the latter and produce ptosis of organs so closely associated. The descent of the intestines is a natural accompaniment.

A tumor of the pylorus may cause ptosis of the stomach, and prolapse of the transverse colon and of the other viscera follow.

On the other hand, a severe type of dilatation of the stomach may be followed by ptosis of the organ and then general visceroptosis. These primary types of gastroptosis are not so very frequent.

The transverse mesocolon surrounds the transverse colon and connects it with the back of the abdomen at the spine. The transverse colon is attached to the abdominal surface of the eleventh rib on each side by a fold of peritoneum. As the colon passes across the abdomen it sags somewhat, presenting a slightly concave surface superiorly. Glénard, whom we must justly credit as the first to describe splanchnoptosis as a pathologic entity, believes enteroptosis (ptosis of the transverse colon) to be the starting-point. He thinks

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[1] This combination has been found in cases at the Manhattan State Hospital.

the transverse colon is fastened to the pyloric end of the stomach by a band (ligament) and that the hepatic flexure first sags, followed by the transverse colon, causing thus a sharp flexion at the attachment of the ligament, and a hindrance to the progress of the intestinal contents, with resulting accumulation in the ascending and transverse colon. From the point of stenosis the transverse colon passes downward diagonally across the abdomen as a hard cord-like mass (corde colique transverse).

The sagging of the transverse colon exercises traction on the pylorus and omentum, thus causing descent of the stomach and liver. The descent of the hepatic flexure, he believes, causes traction on the parietal peritoneum and encourages ptosis of the right kidney. The gastro-intestinal tract, he noted, was suspended in the form of loops, six in number, by means of ligaments; and he believed in the possibility of too great a bend at such an acute angle, that it might cause a partial obstruction to the passage of the contents. This might occur at the gastroduodenal, the duodenojejunal, transverse colon, or sigmoidorectal curves.

The gastroduodenal and transverse colon ligaments Glénard holds to be the weakest, and if they give way, with resulting ptosis of the intestine, increased traction and angulation is produced at the next fixation point, causing an enterostenosis.

Glénard found the transverse colon displaced and stenosed in numerous autopsies, and was the first to realize that many cases of so-called nervous dyspepsia were dependent upon these abnormalities.

Riegel has demonstrated that the hepatic flexure is *not dislocated* downward in the majority of cases, and Glénard's explanation I hardly believe tenable, as there are other very important factors which have a bearing. A tumor, however, of the transverse colon or adhesions may produce primary enteroptosis.

We must remember there is *one type of case*, a congenital constitutional defect, the patient with long narrow thorax, who suffers from splanchnoptosis.

The major number of cases of gastroptosis, however, are acquired from various causes; and in my opinion the development of the prolapse of the various organs generally occurs synchronously, the stomach, right kidney, and transverse colon most frequently prolapsing together; while in other cases the left kidney or the rest of the viscera may descend in addition.

These following are the prominent factors which have a marked bearing in preserving the proper position of the viscera:

(1) *The abdominal muscles.*

(2) *The maintenance of normal intra-abdominal pressure.*

(1) *Abdominal Muscles.*—In an interesting article, A Rose[1] calls

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[1] Surgery, Gynecology, and Obstetrics, November, 1906, Physiology and Pathology of the Abdominal Muscles.

attention to the fact that in addition to the usual functions describe in the text-books, in assisting expulsion of the fetus, bowel actio urination, and vomiting, the abdominal muscles aid in the preservation of the physiologic position of the abdominal organs. The crosswise arrangement of the external and internal oblique and transversalis muscles—supported by the recti—effect a narrowing of th abdominal cavity and prevent visceral ptosis.

Groddeck, of Baden-Baden, has, moreover, described the mechanical influence of healthy muscle, by the alternate contraction and expansion, in assisting the circulation of the blood and lymph, an an atonic condition of the abdominal musculature would certainl interfere with the maintenance of the normal relations between th extra- and intra-abdominal circulation. Moreover, clinically, simpl inspection will differentiate between normal conditions and th typic "pot-belly" of the gastroptosis patient. Acute or wastin disease may also cause changes in the muscles.

(2) *Intra-abdominal Pressure.*—Normal abdominal muscles als maintain the normal intra-abdominal pressure necessary to preserv the position of the viscera.

Walkow[1] has made a very exhaustive study of this question an has demonstrated, *for example, on the cadaver,* with the upper par of the trunk elevated (the reversed Trendelenburg position), that after abdominal section mobility, of varying degrees, of the kidney i found, which did not previously exist.

Stürmdorf has found similar results after laparotomy on the living.

Clinically, changes in the intra-abdominal pressure, the result of childbirth or tapping for ascites, have resulted in the production of splanchnoptosis, the thinned and distended musculature of the abdomen also being a factor.

Rapid loss of weight from emaciation and absorption of omental fat is another example.

Nephroptosis.—Movable kidney, in probably 95 per cent. of cases in my own experience, is one of the stigmata of gastroptosis. The congenital type, with long mesonephron, or those cases due to traumatism, are comparatively few in number.

The right kidney has a longer pedicle and lies lower on account of the liver.

Stürmdorf refers to certain skeletal deformities as influencing the shape of the bony receptacle for the kidneys, and which in some cases predisposes to prolapse; but gastroptosis is associated with these same conditions.

It has been claimed that there is a nephrocolic ligament connecting the kidneys to the ascending and descending colon, and that traction of the colon may influence its descent. Reversed peristaltic action occurring intermittently in the ascending colon, which does not take place in the descending, is believed to have an influence, and the peritoneum over the left kidney is said to be thicker. The fact that the tail of the pancreas lies in front *of the left kidney* seems to me to have some bearing on the question. Absorption of the fatty capsule is probably another factor.

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[1] Medical Record, January 13, 1906.

The peculiar position of the right kidney and lessening of intra-abdominal pressure seem to be the chief causes of its more frequent descent.

With gastroptosis we have also a relaxation of the gastro-intestinal musculature and of all the peritoneal ligaments. Changes in the position of the stomach and in its secretory and, at times, in its motor functions account for the gastric disturbances. The secretory function one might expect to be influenced by circulatory disturbances following displacement of the organ.

Associated are changes in the position of the duodenum productive of stasis, and which readily account for gall-bladder symptoms simulating stone, so often attributed to nephroptosis. Similar disturbances in the intestine, constipation, diarrhea, mucous colic, or chronic appendicitis[1] can thus be accounted for. There is a relaxation of the broad ligaments and with it ovarian and uterine descent, and even descent of the pelvic floor, with dysmenorrhea and various pelvic symptoms.

Occasionally Dietl's crisis from torsion of the kidney pedicle and, rarely, nephritis or hydronephrosis occur. More rarely the kidney may become adherent to the gall-bladder or appendix. In addition, circulatory disturbances and marked neurasthenia, the latter due chiefly, I believe, to auto-intoxication, are present; and from the severe type of splanchnoptosis we have the symptoms-complex of Glénard's disease—all of which the "kidney experts" attribute to nephroptosis.

Etiology.—We must remember that the vertical stomach is the fetal position of the organ and every infant is born with it in this position. After a few weeks or months, through the weight of the food and the action of the diaphragm, the position of the stomach becomes normal. Occasionally it may remain vertical, but I believe this is true more especially in those suffering from the congenital constitutional defect, to which I shall refer.

The causes of gastroptosis may be divided into congenital and acquired:

1. Congenital constitutional defect, the long narrow thorax, with the diaphragm and liver pushed down. In these, splanchnoptosis is a constitutional defect. Stiller's floating tenth rib is usually present. 2. Other skeletal deformities, spinal curvature, rickets, kyphosis, kyphoscoliosis. 3. Intrathoracic pressure on the diaphragm from effusions, tumor, etc. 4. Tumors of the liver. 5. Leukemic

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[1] I do not agree with Edebohls' theory of compression of the superior mesenteric vein by the kidney as a cause of congestion of the appendix.

enlargements of the spleen. 6. Tumor of the pylorus or adhesions (gastric). 7. Tumor of the colon or adhesions. 8. Chronic dilatation of the stomach. 9. Compression of the thorax by tight lacing, poor corsets, tight waist-bands, etc. 10. Relaxation of the abdominal muscles and diminution of intra-abdominal pressure. This may result from rapid emaciation in acute disease, with degeneration of the muscular tissue; or the same condition in longer chronic wasting disease, or *from loss of weight and muscular tone from any cause.*

Landau's disease, splanchnoptosis following confinement, is fairly frequent. Emptying of the uterus produces a sudden diminution in intra-abdominal tension, and the tendency of the viscera is to fill the vacuum previously occupied by the uterus. The abdominal walls are lax and thin from uterine pressure. The accoucheur is often to blame for not properly supporting the relaxed abdomen.

Tapping of ascites, with removal of all the fluid, may produce a similar condition; or removal of large tumors.

Sex.—Meynert found in 50 girls, age twelve, 50 per cent. gastroptosis, and about 80 per cent. females in his gynecologic clinic to 5 per cent. males among adults.

From the study of various statistics it can be estimated that from about 20 to 25 per cent. of women complaining of digestive disturbances are affected with movable kidney and enteroptosis. Unquestionably the percentage of gastroptosis among all women, including those who complain of no symptoms, will average at least 15 per cent. in our city population. The advocates of nephropexy find nephroptosis, disregarding the other ptoses, in 20 to 33 per cent. of all women, a satisfactory surgical viewpoint.

The improvement following *promiscuous nephropexy* in gastrointestinal and other symptoms can be often imputed to the postoperative rest in bed and to the increase in fat by proper feeding.

The ratio of males complaining of digestive disturbances, with enteroptosis and nephroptosis, is about 2 to 3 per cent. The ratio in women is 8 or 10 to 1 male.

Glénard finds a lower ratio, 70 women to 30 men in 100 cases.

Age.—The most frequent age is from eighteen to forty, though between fifty to sixty the condition appears most marked.

Symptoms.—Gastroptosis may exist without the production of any symptoms; while, on the other hand, it may be present with those of a mild character, or may finally present the aggravated type of Glénard. The following symptoms in part or whole may be present: Some cases complain chiefly of nervous, cardiac, gastrointestinal or pelvic disturbances, or of special organs, such as the kidney or liver.

There is usually anemia, a feeling of weakness or faintness, and fatigue on slight exertion and backache. The appetite in some cases is poor, while in others quite good.

Some patients have the symptoms-complex of hyperchlorhydria, while others complain of belching and discomfort immediately after eating. There is usually marked and obstinate constipation, rarely diarrhea; at times intestinal catarrh or mucous colic. Flatulence is present. They have headache, are frequently nervous and hysteric, and at times neurasthenic. There is often a feeling of weight or bearing down in the abdomen, which is relieved by proper support. Menstrual disorders are frequently present, dysmenorrhea quite often. At times the pain and discomfort are focused in the kidneys, especially the right; and in addition they may have attacks of Dietl's crisis. Pains in the region of the liver and gall-bladder occur in some, and there may be attacks of pain resembling gall-stones and occasional jaundice. There may be irritability of the bladder, with frequent inclination to urinate, and pains in the ovaries and appendical region. Various sensitive points are often found in other regions of the abdomen. Palpitation is frequently present and occasionally attacks of tachycardia may occur.

Physical Examination.—*Inspection.*—These patients are usually thin and slender; the abdominal walls are generally flaccid. There is a concavity between the costal arches in the epigastrium from the ensiform to the umbilicus; and in some a vertical median sulcus between the recti muscles wider above than below.

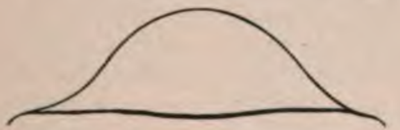

Fig. 153.—Abdominal projection; lead-tape outline between anterior superior spines; curved line, when standing; flat line, when lying on back (from Gallant).

In the dorsal position the abdomen may be flattened below and bulge laterally; and when the patient is erect the epigastrium becomes more depressed; while the hypogastric regions from the umbilicus to the symphysis and the pubic region markedly bulge forward and outward (pot belly). Fig. 153 shows this clearly.

Palpation.—Diastasis (separation of the recti muscles) can be readily appreciated. Stiller's floating tenth rib is present in some cases. Marked pulsation of the abdominal aorta is often met with, as it is uncovered by the stomach. Movable kidney of varying degrees can be readily appreciated, and *this, taken in connection with the splashing sound* found below the normal position of the lower border of the stomach, is pathognomonic of gastroptosis. The corde colique transverse is generally found to be the pancreas, which may also prolapse.

Splashing Sound.—This is the best method to determine the lower border of the stomach and has been thoroughly described.

If no splash can be originally detected, create it artificially by giving water, or if required, add a little Vichy, or tartaric acid and sodium bicarbonate.

Inflation of the stomach with air or CO_2 will settle doubtfu cases, as the upper border is then to be seen on inspection, and percussion is an aid.

Gastrodiaphany is an accurate method.

Percussion.—There is at times dulness or flatness in the epigastrium when the stomach is markedly depressed, the liver descending in such cases. It is difficult to differentiate by simple percussion unless CO_2 distention has also been employed. The scratch method is of assistance.

Gastric Secretion.—*Examination of the Gastric Contents.*—Ewald's test breakfast should be employed and gastric analysis made in every case. Hyperchlorhydria, hypochlorhydria, or, more rarely, achylia gastrica (functional) may be present. Rarely the secretion is normal, and then usually in the cases found accidentally, presenting no symptoms.

I agree with George R. Lockwood to this extent, that in many cases no evidences of fermentation can be found on test, and the gas also may be odorless. In hysteric women some of the air is swallowed. On the other hand, in some patients with associated marked dilatation (the latter probably being primary), such as I have seen among the nervous and insane at the Manhattan State Hospital, marked fermentation has been found.

The treatment is modified by the gastric findings. It is evident, in some cases, that the secretory conditions are influenced by the misplacement, since Graham-Rogers, at the Ward's Island Clinic, found that in 4 out of 7 cases (6 of hyperchlorhydria and 1 of hypochlorhydria) improvement followed the use of Rose's belt[1] alone, without medication or special diet.

Motor Functions.—Motor insufficiency[2] is undoubtedly present in some cases. On the other hand, many cases exist with few or any gastric symptoms, and though there is relaxation of the musculature of the stomach, compensation takes place probably by relaxation of the pyloric ring, so that the contents of the stomach enter the intestine within the normal limit of time. *Per contra* on some occasions, or from some cause, this compensation may fail and then symptoms develop. Motor functions appear only slightly diminished in other cases, or even normal, as just explained.

This is the probable explanation for those cases which have existed for years without symptoms and in whom they suddenly develop.

Stomach and Small Intestine.—Gastroptosis may occur in various forms and degrees, semi-oblique of different degrees, looped, or, more rarely, crescentic, and even the vertical stomach (Figs. 154–158). In some cases we may have primary dilatation and then ptosis. These illustrations represent gastrodiaphany of various cases.

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[1] Rose and Kemp, Atonia Gastrica, p. 124.

[2] Ibid., p. 79.

As far as I can determine, from physical examination, post-mortem, and operative cases, especially in the marked semi-oblique or in the vertical stomach, there seems to be a *straightening of the pyloric curve* and some dilatation of the pyloric end and of the duodenum, probably with relaxation of the pylorus. The supports of the duodenum are relaxed and it crosses the spine at a lower level. I believe there is some relaxation with descent of the pancreas, and also relaxation of the mesentery and descent of the rest of the small intestine.

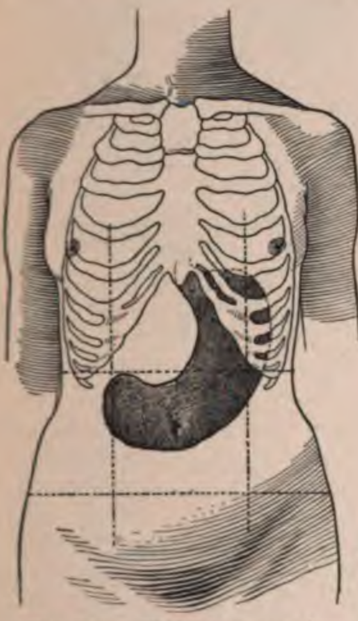

Fig. 154.—Slight gastroptosis.

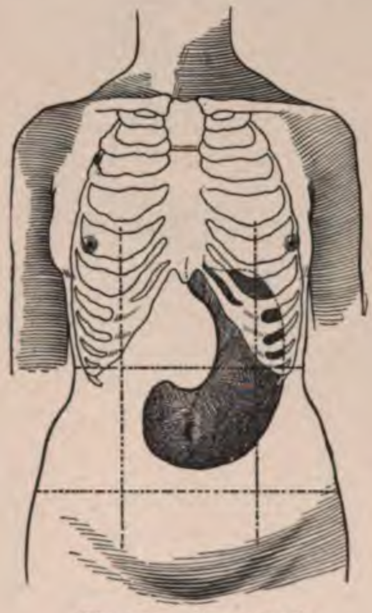

Fig. 155.—Gastroptosis.

The duodenal distention is to be expected from gravitation of the stomach contents, and as there is relaxation of the mesentery I am skeptical of the so-called "chronic mesenteric traction" with dilatation as a result.

Gastroptosis should favor acute dilatation of the stomach, on the mesenteric traction theory, but I have never seen such a condition associated with acute ectasy.

Large Intestine.—*Enteroptosis.*—Descent of the transverse colon is most common (Fig. 159), as can be demonstrated by inflation with air or water, or with bismuth and x-rays; but undoubtedly there may

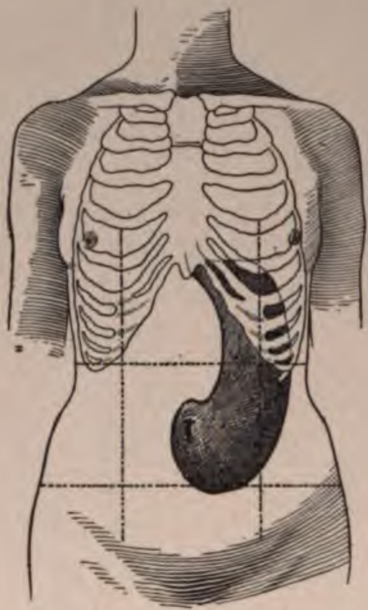

Fig. 156.—Vertical stomach.

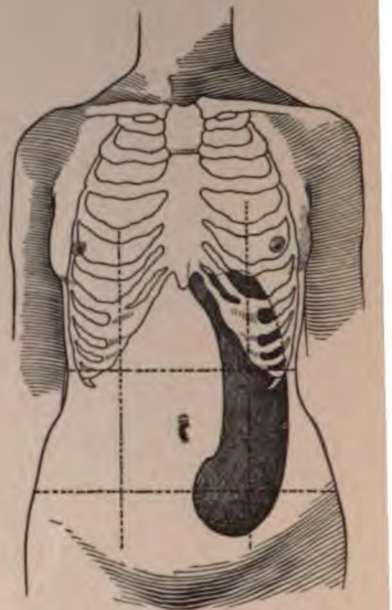

Fig. 157.—Vertical stomach (extreme).

be relaxation, with changes in the position of the sigmoid or even of the descending colon. I have seen a case of this type recently.

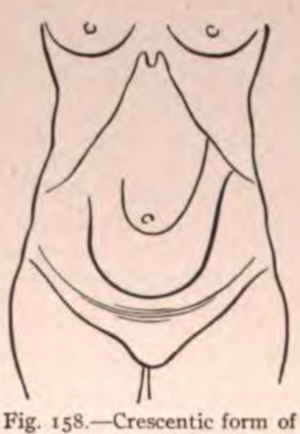

Fig. 158.—Crescentic form of gastroptosis.

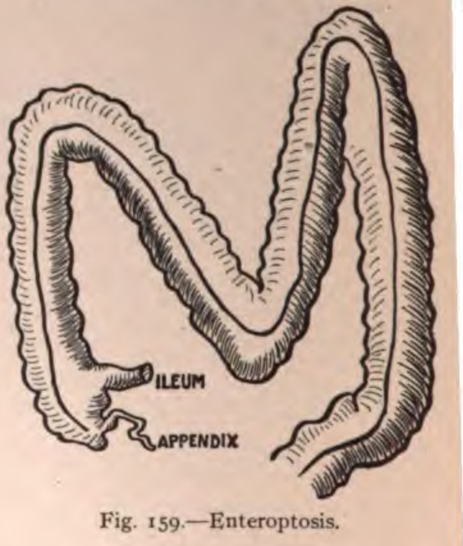

Fig. 159.—Enteroptosis.

Nephroptosis.—It seems important to further discuss this condition, though I have already described many of its features. Movable kidney from traumatism or straining and the congenital floating kidney with a long mesonephron constitute, I believe, a comparatively small percentage of all cases—in my own opinion, about 5 per cent.; while Meynert places them at 10 per cent. The balance are concomitants of enteroptosis (gastroptosis). It is most common on the right side.

Einhorn, as well as many other authors, recognize movable kidney as an essential symptom of enteroptosis.

Nephroptosis occurs at least six or seven times more frequently in women than in men.

Nephroptosis exists in about 15 per cent. of all women examined, associated with gastroptosis; but in many cases no symptoms are present. *No worse judgment can be shown than to tell a patient that she has a movable kidney.*

Edebohls finds it in 20 per cent. of his cases, disregarding associated ptoses, and some even place it at 33 per cent.

The normal kidney is slightly movable during respiratory movements. The most accurate method of kidney palpation I have already described.

Glénard classifies four degrees of movable kidney:

First Degree.—The lower pole of the kidney is palpable on deep inspiration and slips back on expiration. It cannot be arrested.

Second Degree.—The body of the kidney can be palpated and arrested, but not the upper border.

Third Degree.—The superior border of the kidney can be palpated.

Fourth Degree.—The entire kidney is palpable and it may be found in various regions of the abdomen, near the gall-bladder or as low as the appendical region.

I have already referred to the various symptoms attributed to movable kidney, such as dilatation of the stomach due to pressure on the duodenum; jaundice; gall-stone symptoms, or stasis with the production of stone; gastro-intestinal and pelvic disturbances; chronic appendicitis as a result of congestion from compression of the superior mesenteric vein against the pancreas and spine (in 20 per cent. of cases, according to Edebohls).

All these symptoms are referable to splanchnoptosis.

Goelet believes that in 75 per cent. of nephroptosis of the third degree, or beyond, there is a pyelonephritis or interstitial nephritis.

In 20 cases of nephroptosis of the third degree or more (with enteroptosis) at the Manhattan State Hospital, I requested LeRoy Broun to examine the gynecologic conditions, and in no case could any connection be found between the prolapsed kidney and the genital organs. Ferd C. Valentine[1] and Terry M. Townsend, as a check, made special examinations of the genito-urinary tract and urine, but found no evidence of nephritis or pyelonephritis in these 20 cases.

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[1] Kemp, American Journal of Urology, January, 1906.

Such examinations *should not* be made immediately after palpation, as Menge demonstrated that albuminuria appeared directly thereafter.

Schreiber[1] showed this in 39 out of 42 cases examined within ten to fifteen minutes after palpation, and that it *sometimes lasted hours*. Renal epithelium, red and white cells, but no casts were found. He believes it will furnish a *method of differential diagnosis*, as if, on palpation of a doubtful mass, this condition occurs, it will prove to be kidney. Of course, nephritis or pyelonephritis may occur in some cases; and after differential ureteral catherization, if both kidneys are prolapsed, nephropexy is indicated in the diseased prolapsed organ.

Hydronephrosis, or adhesion of the kidney to the appendix or gall-bladder, may also occur occasionally.

In some cases more marked or severe symptoms may be *attributed to the movable kidney:*

(1) There may be a weight or special feeling of traction on that side, increased on standing or walking, and lessened in the recumbent posture.

(2) The kidney may be increased in size, tender on pressure, and there may be pain and tenderness in the lumbar region, frequent urination, and burning headache.

(3) Dietl's crisis, probably due to torsion of the pedicle, will produce severe abdominal pain, chills, nausea, vomiting, fever, and even collapse. The urine may be high colored and blood be present. Abdominal support should first be tried systematically in these conditions before operation is advocated.

Floating Liver (Movable Liver).—Osler claims that in a considerable number of cases *there is a mistaken diagnosis*. One anomaly is the tilting forward of the organ, so that the antero-posterior axis becomes vertical and not horizontal, and a considerable part of the surface of the right lobe is in contact with the abdominal wall.

In one type of lacing liver, the anterior part of the right lobe is greatly prolonged, a shallow transverse groove separating it from the rest of the organ.

A slight grade of mobility (floating liver) is found in enteroptosis, but the cases reported are comparatively few.

In some cases the upper surface may lie below the costal margin. G. E. Graham has collected 70 cases. The condition is rare in men.

In some cases of enteroptosis with hepatoptosis, the symptoms may be fairly marked in the liver, there being pains in the hepatic region radiating toward the back and at times of a tearing character. There may be some local tenderness and attacks similar to hepatic colic, though usually no jaundice.

Einhorn[2] described several groups of cases, dyspeptic asthma among them, but from my own point of view this condition seems to be a part and parcel of the general syndrome "enteroptosis," with marked local symptoms, such as pain or colic, in some cases. In extreme cases the liver may drop down so that the upper surface is below the costal margin.

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[1] Zeitschrift für klin. Med., vol. lv, No. 3.

[2] Medical Record, September 16, 1894.

Cardioptosis.—In my own experience, ptosis of the heart is present only in marked cases of splanchnoptosis, especially where there is skeletal deformity. Einhorn finds it associated with floating liver. The latter is more frequently a concomitant of skeletal deformity, I believe, and hence the two conditions are associated. Movable spleen may occur from below the rib even into the pelvis. There may be dragging pains in the side, torsion of the pedicle, swelling of the organ, with pain and fever associated.

Diagnosis.—Curiously enough, the majority of writers pay chief attention to nephroptosis, refer to the stomach as dilated (a result of kidney pressure), and do not differentiate between gastroptosis and dilatation of the stomach. It is the position of the upper border which determines ptosis, but the following symptoms will usually settle the diagnosis. The peculiar conformation of the abdomen heretofore described, the separation of the recti, *movable kidney, and the determination that the lower border of the stomach is abnormally low*, are sufficiently diagnostic of gastroptosis.

Inflation or gastrodiaphany may be necessary in doubtful cases to settle the question.

Glénard's "Belt Test."—If one stand behind the patient and, encircling him with the arms, lift up and support the lower protuberant abdomen, and this gives relief, it suggests enteroptosis.

Prognosis.—The cases of congenital type with funnel thorax or other skeletal deformities are the most unfavorable to treat; but even in these much can be done.

The acquired type presents a favorable prognosis as to absolute cure, though some tend to relapse if continuous care, as to regulation of mode of life, exercise, and diet is not kept up. I have seen a number of cures, as has every other observer.

Treatment.—*Prophylaxis.*—Much can be done to prevent the acquired type of gastroptosis. Improperly made corsets compress the thorax or waist. Tight lacing and the use of tight bands around the waist are productive of ptosis. These should be avoided.

In my opinion, most of the Landau (post-partum) cases of splanchnoptosis are absolutely preventable, the fault frequently being due to the physician in attendance, the patient's abdomen not being properly supported, and she also being allowed to leave the bed too soon. There is a marked thinning and weakening of the abdominal wall during pregnancy, and after delivery immediate attention should be given to its support.

For several years Douglas H. Stewart, of New York, has been employing as a routine post-partum method of support (at my suggestion) Rose's plaster strapping, reinforcing it with lateral soft-rubber strips (an idea of his own). He states that it supports the organs perfectly, prevents abdominal relaxation, and enables the patient to sit up in bed earlier, with resulting improved drainage of the uterus, and to be about at an earlier period.

Bassler[1] treats of the necessity of abdominal support after confinement, and describes methods of strengthening the abdominal muscles and the value of forced feeding.

Treatment of Existing Gastroptosis (Splanchnoptosis).—There are three chief principles involved:

(1) The support and strengthening of the abdominal muscles, which also *increase intra-abdominal pressure.*

(2) The increase of intra-abdominal pressure, by reducing the volume of the abdominal cavity through accumulation of fat, and thus lifting up the stomach.

(3) Correction of the gastro-intestinal disturbances and the toning up of the nervous system.

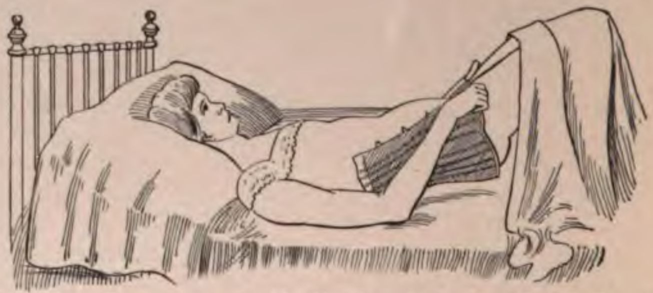

Fig. 160.—The proper way of adjusting the corset (after Gallant).

(1) *Abdominal Support.*—The selection of a proper apparatus for mechanical support to the abdominal muscles, which at the same time will increase intra-abdominal tension, is the first indication.

I have employed, by preference for some time past, adhesive plaster strapping in the form of the belt devised first by Achilles Rose. I have already referred to the superiority of moleskin, first suggested in this country by me after numerous experiments. The Z. O. type should be used by the method already described. The support afforded is continuous, should never be kept on over three to five weeks, and on signs of loosening, a new belt should be applied; the patient, between belts, taking a full bath and employing talcum powder over the surface during the twelve to twenty-four hours' intermission. My longest case wore the belt fourteen months, winter and summer, it being reapplied every four to six weeks, with a gain of 44 pounds and perfect cure.

Some of the other types of adhesive strapping belts may be employed if the wide plaster necessary for Rose's belt cannot be procured. Pressure is exerted by the belt, from the symphysis to the umbilicus in front, the intestines are forced up, and hence the stomach; and the increased pressure aids in holding back the kidneys.

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[1] Prophylactic Measures Against the Development of Landau Cases of Visceroptosis, Therapeutic Gazette, September 15, 1907.

Next in value to adhesive plaster is the Gallant corset, which also exercises upward pressure and support below, and is loose above. The method of its application is shown in Fig. 160.

The La Grecque corset (Von Noorden's), recently devised, is an excellent appliance. It well supports the spine and pelvis, is made in a single piece behind, and is subdivided in front, as in Fig. 161, *A*.

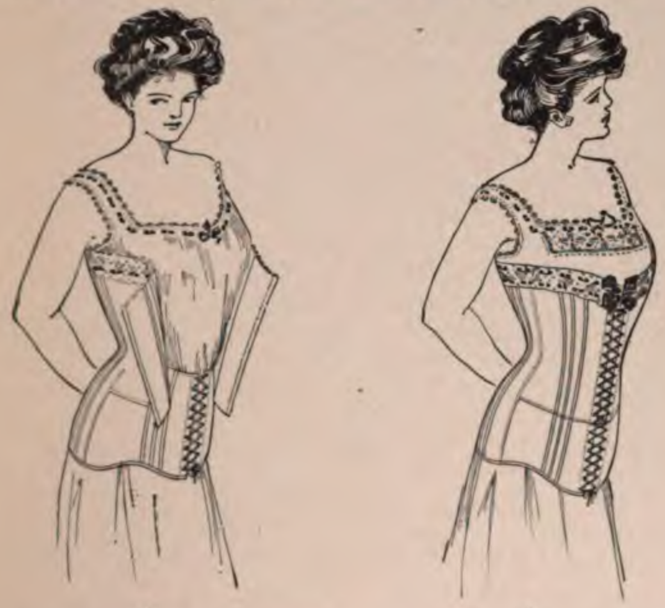

Fig. 161.—La Grecque corset: *A*, Lower segment of corset; *B*, corset after adjustment.

In Fig. 161, *B*, is shown the corset after application. It should be applied in the dorsal position, like the Gallant corset. The pressure is exerted like Rose's belt, from the symphysis to the umbilicus, and it is loose about the thorax and upper abdomen. Of late I use the adhesive strapping for several months and then follow it with this corset. The indications are the same as for Rose's plaster.

If the patient will not consent to these methods, the silk abdominal bandages, as previously illustrated, are useful. In male cases Rose's belt and, later, the silk belt are indicated.

Supports formed with pads for special organs are unscientific.

Kilmer's belt, drawn snug below and lax above, or the Van Valzah-Hayes belt can be employed.

Exercise.—Massage.—Gymnastics.—There are marked inconsistencies in many of the recommendations as to active exercise, flexion, and extension of the body by elevation of the legs, gymnastics, etc., to strengthen the abdominal muscles. There is no better way to take off abdominal fat and to reduce omental fat than by these means; and in the cases with marked weight reduction it is impossible to put on fat if excessive exercises are employed.

In moderation and properly directed, they are of service later when the weight has been increased. Driving or moderate walks in some cases are useful.

Massage.—On the other hand, gentle abdominal massage and often general massage of mild type, taken several times a week, will improve the muscular tone. The use of light cannon-ball massage of the abdomen, taken five minutes once or twice a day, or mild vibratory massage, at home in bed, with the Vedee vibrator, I have found of service.

In the severe type of case, absolute rest in bed for three to six weeks with the Rose's belt applied, forced feeding, and mild massage, etc., give the best results; and if the patient is very much prostrated I omit massage at first.

2. *Increase of Abdominal Pressure through Diet by Fat Accumulation.—Diet.*—There are certain general principles we must follow:

If there is hyperchlorhydria, plenty of albuminous foods and fats and diminished starchy foods should be given; frequent feedings; avoidance of acids, spices, and alcohol. Often alkalis are required.

If hypochlorhydria or achylia, little meat and abundance of carbohydrates and fats, with dilute hydrochloric acid, stomachics, etc.

Not more than 8 oz. (250 cc.) of fluid should be taken at a time, and thorough mastication of the food should be enjoined. If the case is not confined to bed, a rest of fifteen to thirty minutes after each meal, if possible, is advisable. The patient should take the three chief meals daily at—

$$\left.\begin{array}{l}\text{8.00 A. M.,}\\ \text{1.00 P. M.,}\\ \text{6.30 P. M.,}\end{array}\right\}\text{ with intermediate feedings at }\left\{\begin{array}{l}\text{10.30 A. M.,}\\ \text{3.30 P. M.}\\ \quad\text{and often at}\\ \text{9.30 P. M.}\end{array}\right.$$

John Russell's method, as employed in tuberculosis, is of value in some cases. The foods especially of use to increase weight and which can be employed for the interval feedings are:

Milk, raw eggs, cream, ℥j (30 cc.) in milk, ℥viij (250 cc.) kumyss, matzoon, bacillac, lactone-buttermilk, crackers or bread with plenty of butter, and sanatogen (preferably flavored).

Raw eggs can be given, beaten up in milk. It is well to start with 1 or 2 daily, and increase gradually to 6 or 8 per day. They possess considerable value. One could give, for example, at the intermediate feedings:

10.30 A. M.—Milk, ℥viij (250 cc.), with 2 raw eggs.
3.30 P. M.—Kumyss, ℥viij (250 cc.).
9.30 P. M.—Same as at 3.30 P. M., and vary the methods.

Two soft-boiled eggs can be given for breakfast.

Green vegetables and raw or cooked fruits should be given for constipation, depending on the gastric conditions. Strict attention to the bowels is imperative.

I have seen the stomach elevated 3 or 4 inches by the increase of fat.

Electricity.—In the ambulant cases the use of static electricity will prove of service with some patients to improve the general muscular tone, or it may be employed after the rest cure has been completed. The external application of galvanic or faradic electricity I have used with good results in some cases, but never remove the belt.

Intragastric faradization has been recommended to improve the atony of the stomach, and galvanization for the pain. I have had patients bitterly complain of the intragastric method; one case notably had been treated thus systematically, with increasing nervous symptoms being produced and marked nausea, stating she was worse after each treatment. The simple method of external application seems to me advisable in those cases.

Massage of the Kidney.—Several methods have been recommended for treatment of the painful movable kidney:

The operator, sitting on the side of the kidney to be massaged, places the left hand (if it is the right kidney) on the lumbar region, so that the organ rests on the finger, the thumb being supported by the ribs. With the finger-tips of the right hand he pushes on the kidney from in front and gently kneads it.

Brandt places the patient in the lithotomy position, and having replaced the kidney, places both hands in front under the margin of the ribs, and makes vibratory movements backward and upward, allowing the finger-tips to slip around to the back. The patient aids in securing this position by lifting his buttocks.

As palpation *alone will cause albuminuria, I do not approve of these methods, but merely refer to them.* It is sufficient to replace the kidney.

Elevation of the Foot of the Bed.—This method may be employed in those cases taking the rest cure who do not wear an abdominal support continuously. Elevation of the buttocks may be substituted. They are unnecessary when Rose's belt is employed.

Iron and arsenic are indicated in all cases, such as—

Iron tropon, ʒj (4.0), t. i. d.; or
Peptomangan (Gude), ʒj to ij (4.0–2.0); with
Fowler's solution of arsenic, ♏3 to 5 (0.177–0.296 cc.), t. i. d.
or any good combination of iron and arsenic.

An excellent pill is Blaud's iron, gr. 5 (0.3), in which sodium arsenate, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021), has been incorporated.

If no hyperchlorhydria is present, strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021), can be included.

Hyperacidity should be treated with the alkalis.

Hypo-acidity with nux, strychnin, hydrochloric acid, and stomachics.

Bromid, gr. 10 (0.6), or veronal, gr. 5 to $7\frac{1}{2}$ (0.33–0.5), sulfonal, gr. 10 (0.6), or trional, gr. 10 (0.6), are valuable for sleeplessness, given an hour before retiring.

Hydrotherapy.—The Scotch douche, one to two minutes over the abdomen, in convalescent cases has rendered service.

The Priessnitz compress aids to relieve pain.

A glass of cold water, Vichy or Carlsbad, on rising helps the bowel action.

Dietl's Crisis.—Apply heat to the kidney; employ fluid diet; elevate the foot of the bed; replace the kidney.

Codein, gr. $\frac{1}{4}$ to $\frac{1}{3}$ (0.016–0.022), or morphin, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), may be required, by hypodermic.

Constipation.—Olive oil enemata; cascara; comp. rhubarb, if required, are excellent.

Complications,[1] such as mucous colic or catarrhal colitis, must be appropriately treated.

Surgery.—In splanchnoptosis, if all medical treatment after a year conscientiously applied prove a failure, ***suture*** of the recti (abdominis) and thus tightening the abdomen, as advocated by Robert T. Morris and by Charles Codman,[2] is the most scientific procedure.

If there is nephritis, pyelonephritis, or intermittent hydronephrosis confined to the prolapsed kidney, then nephropexy is indicated. After repeated attacks of Dietl's crisis, when medical measures afford no relief, nephropexy may be advised.

Hydronephrosis or pyelonephritis may be of such a character as to require nephrectomy.

Operations on individual organs *I am opposed to* unless there be marked gastric dilatation *with the ptosis.*

Gastroplication, shortening of the lesser omentum, attaching the lesser curve of the stomach to the abdominal wall, suture of the transverse colon to the abdominal wall, sewing the greater omentum to the abdominal wall, have all been recommended.

Gastro-enterostomy may be indicated in gastroptosis with ectasia and marked fermentation, which does not respond to medical treatment. The spleen has been stitched up or removed if inflamed.

Duodeno-enterostomy has been advocated by Byron Robinson, but I see no indication for it.

Elliot[3] recommends anchoring the liver in hepatoptosis.

In *rare cases*, gastroplication, with shortening of the lesser omentum; or the latter with gastro-enterostomy might be necessary.

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[1] See under Constipation for other remedies.

[2] Medical Record, October 19, 1907.

[3] Medical News, November 12, 1904.

CHAPTER XVII

NERVOUS AFFECTIONS OF THE STOMACH

Gastric neuroses may be defined as functional disturbances of the stomach without any discoverable anatomic basis, there being no organic lesion of the organ.

Etiology.—These cases have either inherited a nervous constitution or, through indiscretions, have brought about a condition of nervous prostration. Sometimes the gastric disturbances have apparently a reflex origin, depending on disturbances in remote parts of the body, as in the sexual organs, appendix, eyes, etc., and these factors must always be searched for.

They are divided into sensory, motor, and secretory neuroses, and may appear separately or in combination. They occur most frequently in women from puberty to menopause, and quite frequently at these special periods. In men they appear most often during middle life and most frequently in the higher classes. There is probably a predisposition to the condition. Worry, extreme mental exertion, excesses, and excitement may be predisposing factors. Organic lesions of the stomach must be excluded.

Classification.—Neurosis of the stomach may present the appearance of some primary disease, or may be one of the symptoms of hysteria or neurasthenia, or may *be a reflex symptom due to disease of some other organ.* In cases of this last description, though there is no organic change in the stomach and the disturbance in this organ is purely reflex, there is an anatomic cause elsewhere. They should, strictly speaking, be differentiated from the pure gastric neuroses, since treatment of a distant diseased organ may cure an apparently pure gastric neurosis. This emphasizes the necessity of thorough examination.

Peculiarities.—Generally the nervous system shows more or less deviation from normal conditions. Leube and Boas have probably best described the condition. There are headache, mental depression, lack of energy; at times fear, palpitation, dyspnea, and sweating. On the other hand, there may be excitement and sensitiveness. There are an increase or diminution of reflexes, local hyperesthesia, paresthesia, or anesthesia. Polyuria may or may not be present. Some patients remain in comparatively good condition, while others emaciate.

The digestive disturbances are usually *independent of the quantity and character of the food*, and are not always connected with the act of digestion. Errors in diet are often not followed by an exacerbation of the symptoms, and sometimes they occur when the simplest food has been taken.

The gastric secretion and motor functions are often variable. We may find hyperchlorhydria at one time and normal secretion at another, or, conversely, normal conditions may alternate with hypo-acidity or anacidity. At times the motor functions may be normal, at others, diminished.

The condition of the bowels also varies; there may be constipation alternating with diarrhea; or the bowels may be regular and a sudden diarrhea appear.

Climate and surroundings may influence the condition.

SENSORY NEUROSES OF THE STOMACH

There may be abnormal sensations more or less general in character *external to the stomach*, such as regarding hunger, appetite, or abnormal sensations within the stomach itself.

Among the abnormal sensations of appetite are bulimia (or canine hunger); parorexia (perversion of appetite); polyphagia (excessive eating); akoria (absence of satiation); anorexia (loss of appetite).

BULIMIA

Bulimia, hyperorexia, or canine hunger (cynorexia) denotes a marked increase of the sensation of hunger which may occur in attacks, either as an independent neurosis or as a secondary affection, the result of some other disease. The attacks are paroxysmal. The hunger center is believed to be located in the medulla. This center is probably irritated when a certain degree of impoverishment of the blood takes place. On the other hand, if the latter is marked, the sensation of hunger may not be produced or may even be suppressed. The stomach is also concerned in its production, and the amount of food contained therein has an influence. For example, with hyperacidity and increased motor function, hunger is present. In a case of mine suffering from stenosis of the pylorus and dilatation of the stomach, while before operation anorexia was marked, the patient is now continuously hungry. Reflexly, the hunger center is often disturbed; this loss of appetite may occur after fright or on the appearance of food that is greasy and badly served, whereas with food daintily prepared and of pleasant odor, the appetite is increased. The time at which hunger appears depends on the time the person is accustomed to eat.

Pathologically the sensation of hunger may also be stimulated or inhibited in various ways.

Etiology.—Bulimia may be a primary affection or may be secondary to ulcer of the stomach, hyperchlorhydria, hypersecretion, epilepsy, hysteria, neurasthenia, tumor of the brain, tapeworm, intestinal diseases, rarely to diseases of the pancreas or to diabetes mellitus.

Occurrence.—It is more frequent in women than in men and most frequent from eighteen years of age to menopause.

Symptoms.—The patient may be in perfect health, when suddenly a feeling of intense hunger comes on which is extremely persistent and calls for food. There is frequently a gnawing in the stomach and a feeling of fear and anxiety. Unless the hunger is relieved, headache, trembling, and even fainting spells may occur. The attack may take place immediately after a large meal or come on in the night. In some cases small amounts of food will relieve the conditions; in others, enormous quantities are necessary. One patient recorded ate 23 eggs in forty-five minutes and drank 1½ quarts of milk and 1 quart of wine.

Character of Attacks.—They may be severe or very slight. In some cases they occur every few hours; in others they last a few days, or they may exist chronically and last for years. The periodic form is generally more intense. Hypermotility is found in some cases.

Prognosis.—In the secondary form it depends on the primary disease, though sometimes bulimia persists. The more frequent and violent the attacks, the worse the prognosis. Ordinarily it is difficult to give an absolute prognosis. Gastritis, atony, or dilatation may result in some cases.

Treatment.—If bulimia is secondary, the primary disease should be treated, such as hyperchlorhydria, tapeworm, diabetes, or neurasthenic or hysteric symptoms. For the bulimia we should give frequent light meals every two hours. The bromid of sodium, potassium, ammonium, or strontium may be given in gr. xx to ʒss (1.3–2.0) doses two or three times daily; for example:

℞. Sod. bromid.................................... ʒiiss (10.0)
Aq. menth. piperit......................... q. s. ℥iv (125 cc.).—M.
Sig.—Two teaspoonfuls in water t. i. d. after meals.

Codein, opium, and cocain I strongly deprecate, though recommended by some. There is great danger of acquiring the habit.

Tr. belladonna, ♏x (0.59 cc.) t. i. d., is a good substitute.

Arsenic (Fowler's solution), ♏v (0.296 cc.), or sodium arsenate, gr. $\frac{1}{50}$ to $\frac{1}{25}$ (0.0013–0.0026), or arsenous acid, gr. $\frac{1}{100}$ (0.00065) t. i. d., are useful.

Iron can be given, also strychnin, gr. $\frac{1}{60}$ (0.00108), for the nerves.

Change of climate is of value. Food should be carried by those suffering from abnormal hunger for immediate use.

PAROREXIA (PERVERSION OF APPETITE)

Sometimes the appetite is manifested for special or peculiar kinds of food. There are three types:

(1) *Malacia*, a desire for spiced foods, such as for mustard, vinegar, green fruits, etc.

(2) *Pica*, a desire for substances that are not foods, such as earth, chalk, ashes, sand, insects, etc.

(3) *Allotriophagia*, a craving for disgusting and harmful substances, such as fecal matter, pins, etc.

Malacia is met with in disturbances of the stomach and neurasthenia, while the other types occur chiefly in idiots, lunatics, or severe hysteria. The treatment is of the disease which is the cause of the perversion.

POLYPHAGIA

Polyphagia is the demand for large quantities of food *before satiation* occurs. The cases do not feel hungry until the food is *digested*. It may occur as a primary condition in neurotics, or secondary to diseases of the gall-bladder, spleen, diabetes, or brain tumor. The attacks may be of short duration or as a chronic trouble. One case could eat 100 pounds of meat in twenty-four hours.

Disease, if present, should be treated; neurotic conditions should be corrected. The general treatment is the same as of bulimia.

AKORIA

Akoria is absence of satiety. Patients with akoria never feel *satiated*, and never know when to stop eating. It is at times combined with polyphagia. It is met with among the neurasthenic and hysteric. Bromids are of service and the general treatment of the nervous condition.

NERVOUS ANOREXIA (ANOREXIA NERVOSA)

Anorexia is diminution or loss of appetite, with absence of the hunger sense, so that even *aversion to food* may be present.

It occurs in most of the organic as well as in the functional disorders of the stomach, but the nervous type appears as a primary affection.

It has been attributed to a depressed condition of the hunger center or to hyperesthesia of the mucous membrane of the stomach.

Etiology.—Psychic shock, some mental depression, worry, a disagreeable odor, or some nauseating sight may cause *transitory anorexia* in a healthy person.

It may, however, be more persistent in hysteria, neurasthenia, and the psychoses; *morphin* and excessive smoking may produce it. It is more frequent in *women* and may result from anemia or chlorosis.

Symptoms.—There is at first loss of appetite, and the patient begins to eat less. These cases may first have an aversion to meat, and later to bread and butter, vegetables and all solid food, and may finally live only on fluids, sometimes on very small quantities. They often vomit at the sight or smell of food and may, in severe cases, emaciate markedly. They will refuse to increase their nourishment ("siturgy"). The pulse becomes slow and the temperature subnormal; they become pale, cyanotic, and the eyes sunken. Such cases may even terminate fatally. This condition may be mistaken for organic disease or for phthisis, and a careful physical examination and gastric analysis should be made.

Treatment.—The patient should be impressed with the idea that he must take his food in sufficient quantity and eat everything put before him. He should not be questioned as to his desires.

Frequent small meals with kumyss, matzoon, bacillac, lactone-buttermilk, butter, milk, and cream should be given to improve nutrition. Raw eggs are of service; also sanatogen up to ℥j (32.0) daily in divided doses.

The sour-milk products are of value, as auto-intoxication is an important feature in these neurasthenics.

Stomachics, such as tincture of nux vomica, ♏x (0.59 cc.) t. i. d., and fluidextract of condurango, ♏xx (1.184 cc.), are of service. Fluidextract of Peruvian bark, ʒj (4.0) t. i. d., is excellent.

℞. Tr. nucis vomicæ }
Acid hydrochlor. dil. } ....āā ʒij (8.0)
Comp. tinct. cinchona......................... ℥ss (16.0)
Aq. destil.............................. q. s. ℥iv (125.0).—M.
Sig.—ʒj to ij (4.0–8.0) t. i. d. in water before meals.

Basic orexin, gr. $\frac{1}{3}$ to $\frac{1}{2}$ (0.02–0.03) t. i. d., may prove of service. Morphin and tobacco, if they are factors, should be cut off.

Sanitarium treatment and *rest cure* are important in severe cases, especially by the Weir-Mitchell method. Isolation from the family, strict supervision by the physician, massage, and electricity are valuable.

Forced feeding (gavage) or nutritive enemata may be required. After a while the patient will be convinced she can digest food. Small quantities are given at first, and then they are increased, In milder cases change of climate is useful.

Strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.001–0.002), Blaud's iron pills, peptomangan (Gude), or iron tropon are of service. Arsenic may be added, Fowler's solution, ♏v (0.29), sodium arsenate, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.001–0.002), glycerophosphates of soda, or Chapoteaut's glycerophosphate of lime. These remedies should be given t. i. d.

SENSATIONS WITHIN THE STOMACH

Under *normal conditions* we do not recognize that we have a stomach, and there are no sensations after the ingestion of food. The stomach is not devoid of sensation even normally, as excessive quantities of hot or cold material are noted by internal sensations, such as after the ingestion of a large quantity of ice cream on an empty stomach.

Gastric sensation, however, may be increased to a pathologic degree and be a source of great discomfort. We must remember that certain persons are peculiarly constituted and have idiosyncrasies to particular foods.

Idiosyncrasies.—For example, shell-fish, such as lobsters, crabs, and oysters, strawberries, bananas, onions, etc., will produce skin eruptions such as urticaria or erythema, and gastric symptoms such as pain, belching, pressure, and even nausea or vomiting. Talma and Einhorn have described cases having a peculiar idiosyncrasy to hydrochloric acid. I have seen several cases where the patient suffered from the symptoms of hyperchlorhydria, and yet the free hydrochloric acid found was well within normal limits. The administration of alkalis relieved the symptoms. The gastric nerves were evidently peculiarly sensitive to acid in these cases. If patients have an idiosyncrasy to special food, it must be avoided.

ABNORMAL SENSATIONS (SENSORY NEUROSES OF THE STOMACH)

The nervous and hysteric at times complain of sensations of heat, cold, or of some foreign body, such as the feeling of worms, etc., within the stomach. These symptoms are not dependent on the food or upon the gastric secretion. There may be in some a feeling of constriction, cramp, or "pulsation" within the stomach. The latter is probably due in many cases to hyperesthesia and the transmission of the aortic pulsation.

Nausea.—There may be a nervous type of nausea in such patients, either during the fasting condition or after meals, the treatment for which should be directed to the neurasthenia. Nausea may also be reflex, as from an affection of the genital organs or in organic disease of the stomach. Einhorn recommends intragastric galvanization.

The indication, however, is to treat the hysteria or neurasthenia.

HYPERESTHESIA OF THE STOMACH

This consists in an increased sensibility of the nerves of the stomach, so that the mucous membrane is abnormally sensitive even after ordinary food is taken. It differs only in degree from gastralgia. It may be secondary to organic affections, associated with secretory disturbances, or it occurs as a primary neurosis.

Etiology.—It is present especially in the higher classes. Mental overexertion, excitement, alcohol, and venereal excesses, which weaken the constitution, are factors. It is associated with hysteric or nervous symptoms. Though anemia is given as a cause, hyperchlorhydria with hyperesthesia occur with this condition, and this type is, therefore, not a pure sensory neurosis.

Symptoms.—There is a sensation of fulness, weight, or pressure after meals, which may become real pain, and finally vomiting may occur. Sometimes considerable emaciation is present, as the patient fears to eat (sitophobia). Hyperesthesia occurs after excess of food or, in some, in cases of fasting.

Physical Signs.—The epigastrium and region of the stomach are sensitive to pressure throughout, but there is no specially sensitive area as in ulcer.

Certain articles of food, such as sugar, fat, starch, or coffee, may produce the condition. The secretory and motor functions are normal in the purely nervous cases.

Diagnosis.—This is based on the absence of organic disease of the stomach, the absence of hyperchlorhydria or of other secretory disturbances, and the absence of motor disturbances. With hyperchlorhydria and gastritis the gastric analysis and other symptoms will give us the requisite differential points. With ulcer, hyperacidity is the rule, and the degree of pain is dependent considerably on the character of the food, which is not *generally so in hyperesthesia;* the other symptoms also differ.

With *erosions* small bits of mucous membrane are washed out and there are secretory changes.

Prognosis depends on the result of the treatment of the nervous condition.

Treatment.—In many of these cases, as when anemia is present, secretory disturbances are associated with the sensory neurosis, as hyperchlorhydria, so that the pure sensory condition is often complicated. Iron and arsenic are necessary in such cases and, in fact, have an excellent effect. Peptomangan, iron tropon, or any good iron preparation combined with Fowler's solution, ♏v (0.296 cc.) t. i. d., is excellent.

The patient should be kept in bed in severe cases and warmth applied to the stomach, dry heat, or Priessnitz compresses. Fluid diet in rather small quantity should first be given. Milk and lime-water, sanatogen, broths, chicken soup, white of egg, later entire raw eggs beaten in water, calves'-foot jelly, scraped meat, zwieback softened in milk, butter, and gradually solid food.

Tincture of belladonna, ♏v to x (0.296–0.592) t. i. d., is excellent for the pain. The use of opium and its derivatives and cocain are to be *deprecated.* Rarely an occasional dose of codein may be required.

In some cases silver nitrate, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), in aqueous solution t. i. d. before meals is of service.

External galvanization to the stomach is of value. Bromids in some cases are serviceable.

Baths and change of scene may be enjoined. Suggestion should be used by the physician.

GASTRALGIA

(*Synonyms.*—Gastrodynia, Spasm of the Stomach (Gastrospasmus); Cardialgia, Neuralgia of the Stomach.)

Gastralgia may be defined as the occurrence of violent attacks of pain in the stomach, paroxysmal in character and alternating with free intervals.

Etiology.—We may have a number of forms of gastralgia originating from various conditions, and these must be referred to in order to differentiate them from the purely nervous type.

Gastralgia Originating in the Stomach.—(*a*) Connected with organic affections, such as ulcer, cancer, stenosis of the pylorus, or gastritis, with hyperacidity, hypersecretion, peritonitic adhesions, or perigastritis.

(*b*) Special varieties of food, as rich spices, or of drink, as strong coffee, or ice cream, etc., may produce it in people not accustomed to these substances.

Spinal disease, as tabes, may cause gastric crises, or, more rarely, cerebral disease or various types of myelitis. Absence of patellar reflex, Argyll-Robertson pupil, and Rhomberg symptom are diagnostic of tabes.

Gastralgia as a Neurosis.—This occurs with neurasthenia or hysteria. It may appear before the nervous symptoms are in evidence.

Reflex Causes.—Gastralgia of this type occurs most frequently in women. It takes place reflexly from disturbances in the female or in the male genital organs. Gastralgia may occur at the time of menstruation or in place of it.

Disease of the liver, spleen, bladder, pancreas, and ptosis of abdominal organs may be reflex causes.

Other Causes.—*Malarial infection* may be a cause of gastralgia. The latter may be associated with the usual symptoms of malaria or it may be substituted for the malarial cycle, appearing every day, alternate day, or third day at the same hour, as do other neuralgias.

Gouty infection, lead- or mercurial poisoning, or excessive smoking may produce it. Exophthalmic goiter, anemia, or chlorosis with malnutrition may be a cause, although the attacks in many of these cases are the result of the associated hyperchlorhydria.

Sex.—Most frequently in women and girls from fifteen to forty, and decreases in frequency with age.

Symptoms.—The attacks usually appear suddenly and occur in paroxysms, though they may be preceded by nausea, with belching and distention, headache, or dizziness. There is a sudden extreme pain in the epigastric region of a boring, tearing, burning, and constricting character. It may radiate over the abdomen or to the back and shoulders. The attacks may occur independently of eating and whether the stomach is empty or full, or at any hour (*in the nervous cases*).

The face is pale and distorted with the pain, by which the patient is frequently doubled up, as with colic, and there is inability to lie straight, and often clammy sweating. Strong pressure on the abdomen frequently relieves the pain, though it may be sensitive to lighter pressure. The gastric region is usually sunken.

There are often hiccough and belching, with nausea and collapse. The pulse is generally rapid and feeble, though in some cases slow.

Duration.—Gastralgic attacks may last from a few minutes to an hour or longer. There may be several attacks in a day, every few days, or at intervals of weeks or months.

The pain disappears suddenly and may be followed by hunger or even bulimia. In mild cases the patient may not be greatly affected and may be able to work immediately, while in the severe cases there is often prostration for several days.

Prognosis is favorable as far as life, and depends on the removal of the primary cause.

Differential Diagnosis.—The sudden onset, violent spasmodic pain in the stomach, general in character and lessened by pressure, nausea, vomiting, and headache—that these symptoms *are independent of eating and frequently occur after mental overexertion or emotional shock* and are associated with nervous hysteric symptoms—all point to the nervous type.

We must exclude the secondary form *by gastric analysis*, and by the determination *that no organic disease exists.* A careful physical examination is necessary, thereby eliminating other conditions that may cause pain in the gastric region.

Ulcer of the Stomach.—The pain at the height of digestion depends on the quantity and quality of food; *disappears when the stomach is empty;* intervals free from pain; hyperchlorhydria present; circumscribed spot in the epigastrium painful on pressure and increased by it; hematemesis present in some cases, or occult blood in gastric contents or stool. If these characteristic symptoms are absent it may be necessary to try one of the ulcer cures; which, if it fails, would rather point toward nervous gastralgia.

Cancer of the Stomach.—Pains not as severe, but more continuous, never free from pain; absence of hydrochloric acid; lactic acid and Boas-Oppler bacilli present; cachexia; age of patient; tumor often present.

Chronic Gastritis.—Intense pains are absent; are continuous and more a sense of discomfort after the ingestion of food; no paroxysms; mucus in gastric contents; hypochlorhydria.

Stenosis of the Pylorus.—Attacks of pain (gastralgia), associated with *peristaltic* unrest and vomiting. Dilatation of the stomach is present; usually hyperacid contents if benign stenosis; and lactic acid present with absence of free HCl and Boas-Oppler bacilli if malignant.

Functional Disorders.—*Hyperchlorhydria* and *hypersecretion;* pains disappear after albuminous food or alkalis. Gastric analysis, hyperacidity; and if hypersecretion, excessive quantity of secretion in the empty stomach.

Achylia Gastrica.—Pains disappear when stomach is empty. Gastric analysis shows low acidity, $2+$ or $4+$; free HCl absent; rennet $= 0$; pepsin $= 0$.

Rheumatism and Myalgia.—Myalgia is muscular pain which may be due to exertion. In both conditions the pain is not paroxysmal; corresponds to the course of the muscles, and is relieved by abdominal relaxation; we have rheumatic history or that of overexertion.

Intercostal Neuralgia.—Pain is superficial and can be traced along the intercostal nerves, which are sensitive at numerous points on pressure.

Renal Calculi.—Pain in the kidney (dorsal region) radiates to the ureter and bladder; testicle drawn up in the male; urine is acid and shows blood. Kidney is frequently tender on pressure.

Gall-stones.—Pain over liver and gall-bladder; deep pressure increases the pain; rise of temperature; pain[1] passes to right and often up into the right shoulder. Leukocytosis frequently present.

Jaundice at times and gall-stones in the stool are conclusive.

If the motor or secretory functions of the stomach are normal, the attack of pain—especially if it is in the right hypochondrium and *the patient has no nervous symptoms*—is probably due to gall-stones.

In gastralgia there is no pain and no fever. It is sometimes difficult to differentiate between the other conditions.

Perigastritis.—A high position of the stomach, with the left lobe of the liver drawn down and covering that organ, is suggestive of perigastritis when pains come on regularly several hours after meals (Kaufmann).[2]

Intestinal Colic (*Enteralgia*).—Pain changes its position in the abdominal cavity. Passage of flatus relieves pain. Bowels are irregular.

Treatment.—In secondary gastralgia it is necessary to treat the primary cause.

If it is due to tobacco, it should be cut off. Malaria should be treated with quinin or Warburg; and anemia, with iron and arsenic and the rest cure. Correct a gouty tendency, if present; or treat tabes, if this be found.

Sexual disorders, ulcer, gall-bladder disease, or cancer should receive appropriate treatment.

If hysteria or neurasthenia is present, tonics, isolation, the rest cure, massage, and hydrotherapy are of value.

The galvanic current, the anode over the stomach, or intraventricular galvanization, with the cathode over the spinal column, is of service when repeated attacks of primary gastralgia occur.

For the active condition, if moderately severe, hot applications (dry or moist heat) and hot drinks are useful.

Tincture of belladonna, ♏x to xv (0.592–0.088), to relieve the spasm. Tincture of valerian, ♏xx to ʒss (1.184–2.0), in water, or Hoffmann's anodyne, ʒss (2.0), may be given in water with sugar.

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[1] Head's gall-bladder zone often present.

[2] American Medicine, vol. vi, No. 20, pp. 792, 794, Nov. 14, 1903.

Chloroform spirits, 2 to 3 drops in water at a dose, can be administered. The bromids are also valuable.

If there is retention of food during the attack, then lavage with hot water will relieve some cases.

If the pain is unbearable, codein, gr. $\frac{1}{2}$ (0.032), by hypodermic, or morphin, gr. $\frac{1}{4}$ (0.016), with atropin, gr. $\frac{1}{100}$ (0.00065), can be administered.

Suppositories of morphin, gr. $\frac{1}{4}$ (0.016), and extract of belladonna, gr. $\frac{1}{4}$ (0.016), or opium, gr. 1 (0.065), and extract of belladonna, gr. $\frac{1}{4}$ (0.016), every two to three hours for several doses, are useful.

GASTRALGOKENOSIS (BOAS[1])

This is characterized by pain in the stomach when it becomes empty, and is relieved by food. It may be periodic or permanent. Frequent meals and nerve sedatives are required.

MOTOR NEUROSES OF THE STOMACH

Under normal conditions the cardia remains closed after the process of digestion has begun, while the pylorus opens at intervals to allow a certain amount of escape of chyme. The muscular movements of the stomach mix the ingesta with the gastric juice and aid in its disintegration by churning movements. Later they propel it into the intestine. These motor functions may be irritated (exaggerated) or depressed (diminished).

HYPERMOTILITY OF THE STOMACH

(*Synonyms.*—Supermotility; Hyperkinesis; Hyperanakinesis Ventriculi—Einhorn).

In this condition the stomach propels the ingesta into the intestine more rapidly than normally. It may be secondary to achylia gastrica or hyperchlorhydria or diseases complicated thereby, or the condition may exist as a primary neurosis.

In some cases achylia and hyperchlorhydria exist as a secretory neurosis, and with this there is the motor neurosis, hypermotility.

Supermotility may exist as a motor neurosis alone (primary neurosis), due to some nervous influence or associated with nervous conditions, and is analogous to increased intestinal peristalsis from nervous influence. In pyloric obstruction there is often actual increased motor power in the early stages, but the resistance to the exit is so increased that the final result is relative motor insufficiency. In the secondary cases hypermotility will be relieved by treating the cause.

Diagnosis.—Aspiration of the stomach contents will demonstrate the condition. For example, one may find the stomach empty thirty-five to forty-five minutes after the test breakfast, or only a minute quantity can be removed.

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[1] Boas, Krank. des Magen, II, Feb. 418, Auflage, S. 260, Leipzig, 1901.

Einhorn suggests the employment of the gastrograph, noting an increase of the "makes" and "breaks," but this method is complicated.

Careful analysis of the gastric contents should be made to test the secretory functions, and an early aspiration would be necessary.

Hypermotility in itself does not produce any special gastric symptoms.

PERISTALTIC RESTLESSNESS OF THE STOMACH (KUSSMAUL)

(*Synonyms.*—Peristaltic Unrest; Tormina Ventriculi Nervosa.)

This is really an exaggerated hypermotility in which the peristaltic action is unusually great, and is appreciated by the patient as a disagreeable sensation. It may even become visible through the walls of the abdomen, and waves of contraction can be seen running from left to right along the stomach, and may take from three-quarters of a minute to a minute or more. This condition usually occurs when there is obstruction at the pylorus or duodenum, with dilatation of the stomach, and is produced by the effort of the organ to overcome the obstruction.

Kussmaul first described 2 cases of peristaltic unrest which were pure neuroses of motility. The movements began early in the empty stomach and became more violent after eating. Gastroptosis was present.

Emotional shock and abuse from sexual excesses were the causes.

Mechanical forms must be excluded. In these it occurs only when the stomach is full. In the nervous cases movements take place both when the stomach is full and empty. If the patient is well nourished and the stomach is in normal position it may be difficult or impossible to see the movements, but the patient will complain of the contractions and wave-like movements, and borborygmi and gurgling may be heard. Nausea, vomiting, and cramp-like pains may occur, especially in the obstructive type.

Physical examination and gastric analysis will determine whether the peristaltic restlessness is primary (a neurosis) or secondary.

Course and prognosis depend upon the cause and the nervous condition in primary cases.

Cases may be continuous, when they may cause loss of sleep, or may be intermittent.

Treatment.—This must be directed against the primary cause, such as stenosis of the pylorus, etc.

If it is a pure gastric neurosis, resulting from mental overexertion, grief, sexual excess, or nervous or hysteric conditions, we must remove these factors. Iron, arsenic, and the glycerophosphates are indicated.

Hydrotherapy, diet, and change of scene are valuable. The diet should be regulated, all irritants to the stomach should be avoided. The food should be digestible, non-irritating, and in moderate quantities at a time, especially at night.

Locally, heat or cold to the stomach; galvanic electricity, percutaneous or intragastric. This last is of no value in stenotic cases, but does harm.

Lavage is indicated if there are stenosis and dilatation.

ANTIPERISTALTIC RESTLESSNESS OF THE STOMACH

In rare cases the peristaltic action is reversed, the waves running from right to left. These cases are generally of neurotic origin.

Intestinal waves are of small caliber and move in different directions in various regions, while gastric waves are of large size and move in the gastric region. These waves may extend to the intestines and colored enemata have been voided from the mouth (Osler).

Treatment should be directed to the neurosis.

INCONTINENCE OF THE PYLORUS

(*Synonyms*.—Insufficiency of the Pylorus.)

This condition was first described by de Séré and Ebstein.

It may be caused by some growth in the pylorus, keeping the opening patent, or by stenosis or other organic changes, or when the pylorus is relaxed (atonic) due to some nervous derangement, hysteria, or myelitis.

Relaxed pylorus, I believe, to be frequently associated with atonic ectasy, and that this feature accounts for the absence of pain and vomiting. It is also present in some cases of gastroptosis. The relaxation in these cases is probably compensatory.

Ebstein[1] has shown that if we attempt to inflate the stomach with air or CO_2, it will rapidly pass into the intestine and distend it, and gastric tympany is absent. *A diagnostic feature is the regurgitation of the intestinal contents into the stomach.* On aspiration or lavage in the fasting condition, intestinal juice and often a considerable quantity of bile will be found. It is sometimes present on aspiration of the test breakfast or test meal. *Very little chyme* is present in these cases, but regurgitation is *in evidence.*

There are no distinctive clinical symptoms in most cases; though Knapp[2] reports diarrhea alone or alternating with constipation, as sometimes associated, and also toxemic symptoms, as does Ebstein; and Einhorn refers to 2 cases in which there was associated cardiac relaxation, with belching as the chief symptom. My prolonged investigations among the nervous and insane lead me to absolutely disagree with Knapp that achylia (functional or organic) and insufficiency of the pylorus are confounded. This author believes there is no functional achylia, and with this I entirely disagree.

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[1] The relaxation in these cases I do not believe to be a true incontinence, but merely atony with weakening of the pyloric musculature, with no regurgitation.

[2] Philadelphia Medical Journal, May 24, 1902.

Treatment.—Occasional lavage; strychnin, gr. $\frac{1}{40}$ (0.00146), and belladonna extract, gr. $\frac{1}{6}$ (0.011) t. i. d., with intragastric faradization (every other day).

Massage and *douches* are serviceable.

SPASM OF THE PYLORUS (PYLOROSPASMUS)

Spasm of the pylorus is usually a secondary occurrence, associated with hyperchlorhydria, hypersecretion, ulcer of the pylorus, or in its immediate neighborhood; and in the latter case must be considered a reflex neurosis. It may result from irritation of the stomach by indigestible diet, by food that is too hot or cold, or by strong spices, etc. It is rare with carcinoma with abundant lactic acid.

Bentijac[1] described a case who drank a glass of kerosene by mistake, and who later developed all the symptoms of stenosis of the pylorus and dilatation.

Operation showed the pylorus normal, but spasmodically contracted. Vomiting ceased after operation.

Repeated spasm of the pylorus, in conjunction with hyperacidity or hypersecretion, is undoubtedly at times the cause of gastric dilatation; and in some cases benign stenosis may result from hypertrophy at the pylorus caused by repeated spasm.

Symptoms.—Pain, increased peristalsis of the stomach, and occasionally vomiting are present.

Pure nervous spasm of the pylorus is difficult to diagnose. All perversions of secretion and all mechanical obstacles must be excluded. Nervous pyloric spasm is most likely to be associated with the hysteric or neurasthenic condition.

Treatment.—Belladonna is of value; the tincture, ♏x (0.592) t. i. d., or extract, gr. $\frac{1}{4}$ (0.016) three or four times a day.

The nervous system must be treated. In the secondary cases, secretory perversions and other causes of the condition must receive appropriate treatment.

ATONY OF THE STOMACH

This is a condition in which the muscular action of the stomach is retarded and weakened, and moderate motor insufficiency results. It may be acute or chronic and secondary to other conditions, or as a primary neurosis. It has been already fully described.

HYPANAKINESIS VENTRICULI (EINHORN)

is defined as a condition in which the mechanical function of the stomach is greatly reduced. There are no breaks, or few, in the gastrograph, an instrument to determine the churning movements of the stomach. It seems a minor degree of atony, and is of no special clinical significance.

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[1] Thèse de Paris, 1888.

HYPERANAKINESIS VENTRICULI (EINHORN)

This is an excessive mechanical action of the stomach. It seems to correspond to hypermotility. Excessively active churning movements occur in this condition (Einhorn). It seems difficult to separate it from hypermotility.

SPASM OF THE CARDIA (CARDIOSPASMUS)

Spasm of the cardia consists in a spasmodic contraction of the muscles of the cardia, with the production of pain and difficulty in swallowing (dysphagia). It may be secondary to other diseases (organic disease), reflex from local irritation, or as a primary neurosis, associated with nervous conditions.

Etiology.—It may be produced by the passage of the stomach-tube or after rapid eating, by very hot or cold drinks, and by coarse or hard food.

Among other causes are ulcer or carcinoma of the cardia. Hyperacidity may occasionally cause it, though pyloric spasm is more frequent with this affection. Diverticula, or inflammation of the esophagus, are causes. It may occur in tetanus. It is quite frequent in hysteric or neurasthenic individuals, especially in air swallowers (aërophagia), in whom spasm of the pylorus may also occur with resulting distention of the stomach ("pneumatosis"). Violent psychic shocks may also produce it, so the condition may be a primary neurosis. Fermentative processes in the stomach, with the production of gas, may be a cause; but organic disease is usually associated. *Primary spasm of the cardia cannot produce gastric distention* unless spasm of the pylorus is also present.

Symptoms.—*As a pure neurosis* the attack usually begins suddenly. The patient may be in perfect health, when an acute pain begins in the region of the cardia radiating toward the chest or back. It occurs generally during the ingestion of food, and there is a feeling as if some of it were arrested. There is at times some interference with breathing. Slight dyspnea is present and the respiratory movements are more forcible. There may be regurgitation and the patient feels better thereafter. There is gagging and often vomiting.

The attack can be acute and last only a short time or the condition may become chronic. The dysphagia in these cases, as a rule, increases; and though for a time the patient, by taking a deep inspiration and compressing the thorax while holding his breath, can force down the food, the deglutition of solid food becomes more and more difficult, and finally only liquids can be taken. Emaciation may become quite marked, and after several years atonic dilatation of the esophagus may result similar in character to atonic gastric dilatation from pyloric spasm. Dilatation of the esophagus may also be produced by benign or malignant stenosis at the cardia; or by paralysis of the dilator nerves of the cardia, from paralysis of the esophagus, or by loss of reflex relaxation of the cardia (Einhorn).

Diagnosis.—In acute cases the existence of pain and dysphagia, the delay or absence of the swallowing sound, and the spasmodic resistance at the cardia on insertion of the stomach-tube, which can be overcome by pressure, together with the presence of neurasthenia or hysteria, settle the diagnosis.

In the chronic type the history of the case, the fact that often the resistance to the *large tube* is less than to *the small one in nervous cases*, all aid the diagnosis.

In true stenosis (organic stricture) the small sounds are more readily passed. The method of determining esophageal stricture has been described. If blood appears with the sound, ulceration is at once suggested. Cachexia is suggestive of malignancy, and aspiration of the gastric contents will settle the question. Dilatation of the esophagus can be further determined as follows:

A slice of bread can be dissolved in 200 cc. of water to form a soft mass, and administered, the patient being instructed to employ forcing movements, so that it will enter the stomach. An hour later 200 cc. of water may be given, no forcing movements to be employed. In about three to five minutes an aspirating tube can be passed to the cardia, and if dilatation and spasm exist the water just ingested can be removed, and will be in the same condition as taken. The tube can then be forced on into the stomach and the true gastric contents can be removed. These can be tested. The administration of bismuth with the employment of the x-rays is also of value for the purpose of diagnosis.

Prognosis is good in the acute cases, but as regards complete cure in chronic cases it is not so good, though there is no danger to life.

Treatment.—If the spasm occurs while eating and drinking, this should be performed slowly. The food should be well masticated or should be, preferably, soft in character; and excessively hot or cold food should be avoided.

General tonics should be given. The employment of sounds, left in place for some time, is of value.

Tincture of belladonna, ♏x (0.592), should be given t. i. d. to relieve spasm. The bromids are of service; thus, sodium bromid, gr. 15 to 30 (1.0–2.0) t. i. d.

Opium or chloral should be administered with caution, or not at all.

In the chronic cases soft or liquid food should be given. The patient should bear down to force the food thoroughly by the obstruction. The stomach-tube should be introduced at least once a day to relax the cardia; if there is emaciation, food can be administered through the tube; if the esophagus is dilated, it can be emptied and washed out. Rectal feeding must be enjoined for a time in severe cases. As improvement occurs the diet can be increased.

INSUFFICIENCY OF THE CARDIA

Under this we may classify *eructation, pyrosis, regurgitation, and rumination.*

Eructation (Belching).—Eructation may be defined as the expulsion of gas from the stomach through the mouth.

The condition may be secondary to various conditions of the stomach or may be a *primary neurosis.*

Normal healthy subjects may eructate after drinking carbonated water, or those who eat rapidly without proper mastication, and thus swallow considerable air.

Secondary Cases.—Belching may be associated with acute or chronic gastritis or with fermentative processes in the stomach.

The clinical symptoms, gastric analysis, and fermentation test will render a positive diagnosis.

In fermentative processes the gas is often *malodorous.*

Primary Cases.—Nervous Belching (Eructatio Nervosa).—We have cases in which belching occurs at short intervals, which is independent of the character of the food and clearly of nervous origin. It is noisy, as a rule, and may persist for a considerable period or occur in paroxysms.

The gas consists of atmospheric air which has been swallowed (aërophagia) or aspirated into the stomach.

Oser claims that the stomach acts as an elastic sac, and can aspirate the air without the effort at swallowing, and that it tends to fill after expulsion. Some of the air probably comes from the esophagus and has been swallowed just previous to the act of belching. Bouveret considers it due to *spasm of the pharynx.*

Etiology.—This nervous type of eructation is observed in hysteric women and chiefly in neurasthenics, or after excitement, shock, or mental worry. It frequently occurs in children, often in several in a family.

The belching may last for a long period and only occur during the day, and may be extremely annoying.

Diagnosis.—Absence of fermentation, as shown by the test, the history of nervousness or of shock, and the gastric analysis showing normal secretion render the diagnosis conclusive.

Treatment.—Massage, hydrotherapy, iron and arsenic, change of climate, the faradic current (extraventricular or intragastric), bromids, and suggestion to the patient are of service.

PYROSIS (HEART-BURN)

This consists in the ejection of chyme from the stomach into the esophagus, with which is associated a burning sensation in the epigastrium.

Etiology.—It most frequently occurs with hyperchlorhydria, but has been found with achylia.

It may occur as a neurosis, especially among the hysteric or neurasthenic, with normal gastric contents. It is of mixed type, motor and sensory.

General tonics and electricity are of value in these cases.

REGURGITATION

Regurgitation denotes the condition in which after eating, some of the food ingested, liquid or liquid and solid, rises from the stomach, enters the mouth, and is ejected.

Occasionally small fragments are swallowed again. The act is usually involuntary, though it can be produced by some at will. Early regurgitation tastes of the food, later it is acid. The patients *can suppress it at will*, and in this way it differs from regurgitation due to cardiac stenosis or diverticula.

The sound and chemic analysis also give aid in diagnosis. In some cases, if regurgitation is marked, considerable emaciation may result.

Organic disease, such as stricture of the esophagus, must be excluded; also gastritis.

Etiology.—Patients of nervous or hysteric type develop this condition; and mental worry and nervous strain may be the cause.

Prognosis is good.

Treatment.—The patient should eat slowly and masticate his food. Suggestion is good; also forbid the patient to spit out his food, and tell him to swallow it again.

Massage, faradization (intra- and extragastric); strychnin, gr. $\frac{1}{40}$ (0.0016) t. i. d., and the treatment of the nervous condition are indicated.

RUMINATION

(*Synonyms.*—Merycism, "Chewing the cud.")

In this condition the food is returned from the stomach, without nausea, into the mouth, some time after meals, where it is chewed and swallowed again.

Etiology.—It occurs more frequently in men than in women; and most of the cases reported belong to the higher classes, especially among professional men.

Many of the lower class would not deem the condition abnormal and women would conceal it, so it may be said to belong to all classes. It occurs among hysteric persons, neurasthenics, epileptics, and idiots.

Possibly heredity and, undoubtedly, *imitation* in many cases are factors. Occasionally there may be a pathologic condition of the stomach, with regurgitation, and rumination follows. Several cases have been reported of imitation among children.

Shock, trauma, rapid eating, and emotional disturbances in nervous persons are at times accessory factors.

Gastric Findings.—In some cases hyperacidity has been found, and rumination diminished after this was corrected.

Diminished acidity has been found by Boas, and improvement followed after hydrochloric acid was administered.

On the other hand, achylia gastrica has been present in such cases and also normal secretion.

The *motor functions* are normal in most cases.

Prognosis.—The habit may be corrected in part, but may persist for years, with no impairment of health. In some patients the attacks are periodic.

Treatment.—Any secretory anomaly that exists must be corrected. The treatment should be by mental impression (psychic) on the part of the physician. The patient should be taught that he can readily suppress the condition. Small bits of ice after meals; lavage and gavage have been suggested. The nervousness should be corrected by iron, strychnin, and arsenic.

NERVOUS VOMITING (VOMITUS NERVOSA)

Vomiting is a complex process. There is a contraction of the abdominal muscles and fixation of the diaphragm, with a contraction of the stomach, accompanied by closure of the pylorus and opening of the cardia; reversed peristalsis of the esophagus and closure of the larynx and posterior nares, with resulting expulsion of the stomach contents through the mouth. The vomiting center lies in the medulla near the vagus center.

Vomiting may be due to some organic disease of the stomach or as a result of irritation due to the ingestion of food of abnormal character. Certain intoxications, as from uremia, cholemia, opium, morphin, tobacco, ether, or chloroform, may produce vomiting.

Nervous vomiting is characterized by the absence of these conditions just mentioned, and may be either cerebral or spinal in origin, or due to reflex disturbance, overexertion, emotional shock, anger, fright, or neurasthenia.

Among the cerebral causes are organic disease of the brain and its meninges, concussion, and tumors.

Spinal forms, such as tabes, or occasionally paresis, multiple sclerosis, and subacute myelitis.

Reflex causes, such as from the pharynx, larynx, palate, liver, peritoneum, kidneys, genitals, etc. Juvenile vomiting and the periodic or cyclic vomiting (Leyden), also the cyclic vomiting of children (infants) are included.

Characteristics of Nervous Vomiting.—Stiller calls attention to the following peculiarities:

This type of vomiting seems *to be independent of the character and quality of the food, and occurs generally without any premonitory symptoms.* Sometimes digestible foods are vomited, while indigestible material is well borne. There seems at times to be a power of selection to vomit certain nutrients, and the patients suffer from no inanition. Vomiting may also occur from the empty stomach, and the condition is associated with various nervous symptoms.

Boas has noted that the motor and secretory functions are normal, though Bouveret and Einhorn have observed a diminution or absence of secretory function in some cases.

Treatment.—Tonics. Correct disturbance of secretory functions. Mental impression on the part of the physician is important. Change of scene is at times of value.

PERIODIC VOMITING (VON LEYDEN)

There are several particulars connected with this type of vomiting. As a rule, *no cause can be discovered*, and the attack appears when the patient is in perfect health. The attacks occur at intervals of equal duration. After the attack the patient is immediately restored to health, and remains well until the next one occurs.

They resemble somewhat the gastric crises of tabes, and are similar in suddenness of occurrence to violent attacks of migraine. They seem to be influenced by emotional disturbances. They begin with slight nausea and with a chilly feeling and headache followed by vomiting of the gastric contents, and later bile and mucus streaked with blood.

The vomiting is very persistent, and all food or even small quantities of water are ejected. In some cases there is pain. The patient becomes greatly prostrated, the abdomen sunken, and the face pale. The attack may last from a day to a week or longer. Suddenly the nausea and vomiting cease, the appetite returns, and the patient is rapidly restored to health.

Gastric Juice.—This has been found to be normal in most cases, though Einhorn reports a case of achylia gastrica.

Treatment.—Rest, ice pellets, morphin, gr. ¼ (0.016), by hypodermic, and belladonna tincture, ♏x (0.59), are useful. Cocain I deprecate. Tincture of hyoscyamus, 2 cc. (30 drops), and hot applications during attacks. Oxalate of cerium in grain doses is of value. Between attacks, tonics, change of climate, and hydrotherapy.

CYCLIC VOMITING IN CHILDREN

This is probably due to faulty metabolism; occurs generally in those from two to four years of age, of a nervous type and gouty family history.

There are premonitory symptoms; temperature but seldom over 100.5° F., anorexia, languor, followed by persistent and violent vomiting of food, mucus streaked with blood, and at times bile. Prostration is marked; the gastric contents very acid. Acetone, diacetic acid, and oxybutyric acid are present in the urine, and so it is believed it is a form of acid intoxication; uric acid in the urine is diminished. It is possibly a lithemic manifestation.

The attack resembles migraine in the adult. Antecedents are a neurotic and gouty family history. Carbohydrates in excess seem to have a bearing on its production, according to some; fatigue, excitement, or tonsillitis bring it on.

Prognosis.—Good.

Diagnosis.—Attacks are not brought on by indigestible food; the disease is self-limited, and the child rapidly returns to the normal state. The attacks are repeated.

Treatment.—Calomel to abort it; food and drink should be stopped; enemata and enteroclysis are advisable. Hypodermoclysis may be necessary in severe cases and rectal feeding is indicated.

Alkaline Treatment.—Sodium bicarbonate, gr. 15 to 30 (1.0–2.0), in divided doses may be given.

Limit starchy food. Care must be taken not to tire the child thereafter.

JUVENILE VOMITING

Overwork at school is often the cause. Cardialgia and vomiting occur.

There may be headache, slow pulse, pallor, dilatation of pupils, etc.

Treatment.—Tonics, proper diet, removal from school.

REFLEX VOMITING

Nervous vomiting is frequently reflex in character from disease of almost any organ. Among the causes are disease of the pharynx, elongated uvula, diseases of the abdominal organs, as hydronephrosis, movable kidney, kidney colic, ptosis of the liver or spleen, peritonitis, appendicitis, hernia, and disease of the sexual organs. These conditions should receive appropriate treatment.

The vomiting of pregnancy belongs to this type.

Belladonna tincture, ♏x (0.59), t. i. d.; cerium oxalate, gr. 2 (0.13), t. i. d.; Fowler's solution of arsenic, several doses 1 drop each; bromids, gr. 15 (1.0); codein, gr. ¼ (0.016); or chloral, gr. 3 (0.194), t. i. d.

℞.	Menthol	gr. x (0.6)
	Syrup	℥ij (60.0)
	Aq. destil	q. s. ℥iv (125.0).—M.

Sig.—Two teaspoonfuls t. i. d.

The use of cocain I deprecate. Lavage is temporarily of service.

Abortion may occasionally be required in pregnancy cases. Change of scene may be necessary.

IDIOPATHIC NERVOUS VOMITING

In some hysteric or neurasthenic persons (adults) vomiting will occur after meals without any apparent cause, more frequently in women and without showing the periodic type. Usually part of the meal is vomited. It may continue for a long period. Nutrition is frequently not disturbed. The vomiting may occur so quickly during the act of ingestion of food that it seems as if it did not enter the stomach, but was rejected by the esophagus. Other patients seem normal in this regard.

Occasionally hysteric subjects may vomit blood. In all cases the gastric secretion must be studied, so as to exclude hyperchlorhydria, hypersecretion, etc., and to observe whether vomiting complicates some other disease or is a reflex affection.

Treatment.—Suggestion, the regulation of the mode of life, tonics, such as arsenic and iron, bromids; change of climate; gavage for about two weeks; lavage with 1 : 1000 nitrate of silver, have proved of value.

Faradization, extra-abdominal or intragastric (Einhorn), has been of benefit.

PNEUMATOSIS

Spasm of the cardia, combined with pyloric spasm, may produce pneumatosis (distention of the stomach with air), with a resulting sensation of tension and at times dyspeptic asthma.

Hysteric or neurasthenic symptoms are associated.

Aërophagia (air swallowing) is probably the cause. The upper part of the abdomen is markedly distended and tympanitic, and there is interference with respiration. As a rule, there is no belching.

Diagnosis.—Organic affections of the stomach must be excluded, in which the gas has a foul odor and the contents ferment.

Treatment.—Tonics and bromids are indicated. Aspiration of the stomach in the acute attack, so as to give exit to the air, is the best method. It may be necessary to repeat it.

Extract physostigmatis, gr. $\frac{1}{8}$ (0.008), or eserin, gr. $\frac{1}{100}$ (0.00065), or morphin, gr. $\frac{1}{4}$ (0.016), or tincture of belladonna, ♏x (0.59), or extract of belladonna, gr. $\frac{1}{4}$ (0.016), may be required.

SECRETORY NEUROSES

The secretory function of the stomach is undoubtedly under the direct control of the nervous system. For example, in a hungry dog with a fistula the sight of meat will produce gastric secretion, and in the case of a man with impermeable esophagus, but with a gastric fistula, mastication produced gastric secretion. The vagus has been demonstrated to be the secretory nerve. The stomach itself, however, possesses some secretory power, since after section of the *pneumogastric and sympathetic nerves*, secretion will occur after the application of an irritant.

Hyperchlorhydria, gastrosuccorrhea (hypersecretion), and achylia gastrica may all be functional disorders of secretion, and have been described in special chapters.

Subacidity (hyposecretion, hypochylia gastrica, hypochlorhydria) may be of nervous origin, and must be differentiated from cases occurring with organic disease of the stomach, especially gastric catarrh.

Hydrochloric acid, strychnin, massage, and electricity are of service in these last cases.

In the subacid cases sudden changes in the gastric findings (secretory) are in favor of a neurosis, according to Hemmeter.

Disorders of secretion may accompany other diseases, such as tabes, or spinal lesions.

NERVOUS DYSPEPSIA (LEUBE)

(*Synonym.*—Neurasthenia Gastrica.)

Leube originally described nervous dyspepsia as a disorder of the stomach, characterized by a variety of distressing subjective symptoms during the act of digestion, but in which it was *normal as regards time and chemism.* In effect, he originally considered "nervous dyspepsia" as a neurosis of sensibility. He has more recently extended his definition to other forms. Strictly speaking, nervous dyspepsia is a combined *gastric neurosis* in which the sensory disturbances (subjective symptoms) are the most prominent.

It may be combined with secretory and at times even with motor disturbances.

Gastric Juice.—The findings in the gastric juice are not characteristic. It may be frequently normal; it may occasionally be hyperacid, *more frequently diminished acidity,* or at times there may be variations in the same subject. In long persistent cases atony may be present.

Some authors refer to the presence of enteroptosis or membranous colitis with neurasthenia gastrica. In this connection it is evident that frequently the diagnosis of "nervous dyspepsia" is made, when in reality gastroptosis (enteroptosis) is the basis of the difficulty. With the ptosis of the viscera we have sensory, secretory, and frequently motor disturbances of the stomach, associated with nervous symptoms, but the correction of the ptosis will cure the condition.

In pure nervous dyspepsia *all organic lesions of the stomach* must be excluded and also *ptosis of the viscera.* The stomach *must occupy the normal position.*

Etiology.—Neurasthenia gastrica may occasionally appear as an independent neurosis, but more usually with nervous symptoms, hysteria, or neurasthenia. Grip, pulmonary disease, anemia, chlorosis, malaria, or debilitating conditions predispose to it, as do also reflex irritation from the sexual organs, excessive venery, abuse of alcohol, or tobacco.

This disease occurs more frequently in men than in women, especially among brokers and those subject to worry and mental strain, and usually at the prime of life between thirty and fifty, though it may be present at other periods.

Symptoms.—The patient usually complains of a feeling of fulness or pressure after eating, or even of slight pain or belching, loss or irregularity of appetite, a sleepy feeling, or even weakness or dizziness. The tongue is usually clean. There is marked mental depression, and the patients are nervous and anxious. One *peculiarity is that the quantity and quality* of the food *seem to make little difference* in their *symptoms*. At times indigestible food can be taken without discomfort, while at other times digestible food may produce the symptoms. Occasionally the pain and discomfort are present when the stomach is empty. There is often nausea and occasionally vomiting. Thirst is variable. There is usually tension or fulness of the intestines due to accumulation of gas, which is passed later per rectum. The bowels, as a rule, are constipated, with occasionally an alternating diarrhea. The movement may appear in narrow cylindroids or small balls in some cases.

In addition to the feelings of depression, insomnia, palpitation, headache, and lassitude may be present; also vertigo, impotence, or emissions.

The surroundings and general mood of the patient have a marked influence on the symptoms. The cases are often extremely disagreeable in their "home life." Circumscribed points, sensitive to pressure, have been described as diagnostic; one below the ensiform and the others near the navel, especially to the left, but Ewald has demonstrated "nervous dyspepsia" in which no such points could be found.

Course.—The course of the disease is slow and the symptoms vary; sometimes one symptom being prominent; at another time, another. The mood of the patient has a marked influence and in good company he may forget his trouble. They generally do not suffer much in nutrition, but in cases with insomnia they at times lose considerable weight.

Diagnosis.—The presence of general nervous symptoms, especially of those pointing to the stomach, *without the presence of organic disease of the organ;* the fact that the gastric secretion is often found to be normal, though at times hypochlorhydria and more rarely a mild hyperchlorhydria, and that we frequently obtain variable gastric analyses in the same patient; that there is lack of proportion between the gastric findings and the condition of the digestive organs, as compared with the severe complaints voiced by the patient; that the character of the food, whether digestible or indigestible, seems to make no difference as regards increasing or ameliorating the symptoms, and, finally, that change of scene or the mental condition of the patient has a decided influence on the condition—all these facts point to neurasthenia gastrica.

Differential Diagnosis.—The chief differences are as follows:

Neurasthenia Gastrica.—Character of the food makes no difference as to symptoms. There are sudden changes in the patient's condition; well for a few days, and then marked symptoms; nervous symptoms marked. Gastric secretion often normal, and frequently variable in the same patient at different times.

Chronic Gastritis.—Aggravated by errors in diet; symptoms constant; *mucus in the gastric contents;* reduction in hydrochloric acid is marked as a rule.

Ulcer of the Stomach.—Painful area in the epigastrium, tender on pressure; hematemesis or occult blood, melena, and pain is increased markedly after ingestion of food. The character of the food influences the pain markedly, even in the more obscure cases. Pain remits or disappears.

Cancer.—Age of patient; tumor; cachexia; quality and quantity of the food may not markedly influence the pain; gastric analysis showing absence of free HCl, lactic acid, and Boas-Oppler bacilli present; pain continuous; progressive emaciation.

Treatment.—If there are any sexual disorders which reflexly might affect the nervous condition, these should be treated; sexual excesses or overindulgence in alcohol or tobacco should be checked. Brokers and professional men who have mentally overexerted themselves or are tired with the worry of business cares, lead an irregular life, or who are engaged too actively in social pursuits, if possible, should have a change of scene. Horseback riding, golf, yachting, fishing, shooting, camp life for a few weeks, a pleasure trip, all give excellent results. A short ocean trip south or abroad is of service. In those who cannot afford these methods, the lightening of business and professional cares is important.

Hydrotherapy, massage, electricity, especially by the faradic current, combined with out-of-door life and proper exercise, mild gymnastics, so as not to tire the patient, are of value. *The mental impression* created by the physician *is important.* Static electricity is at times of service. General faradization, the bare feet on one electrode, and the other being passed over the body, is useful (Rockwell).

The diet should be abundant, the patient avoiding highly seasoned food, alcohol, strong coffee, and excessive smoking.

Stomachics should be given if hydrochloric acid is deficient, such as nux vomica, compound tincture of cinchona, dilute hydrochloric acid, etc.

If hyperchlorhydria, then the alkalis should be administered. The patient should secure the proper amount of sleep.

Forced feeding and the Weir-Mitchell rest cure are necessary in severe cases. Milk and raw eggs are of value in reduced nutrition.

Malbranc's gastric douche has been recommended in some cases, and Einhorn suggests the use of his gastric spray. Personally I do not employ such local measures.

The general tone of the patient should be built up by iron, such as peptomangan (Gude's), iron pills, iron tropon, arsenic, and strychnin. Small doses of nux vomica combined with compound tincture of cinchona are excellent to improve the appetite:

℞.	Tr. nucis vomicæ	ʒiij (12.0)
	Comp. tinct. cinchona	℥ss (16.0)
	Aq. destil. q. s.	℥iv (125.0).—M.

Sig.—1 to 2 teaspoonfuls t. i. d. in water before meals.

Basic orexin, 2 decigrams (gr. 3) t. i. d., has also been recommended by Einhorn for this purpose.

Sodium or ammonium bromid, gr. v to x (0.3–0.6) two or three times a day, lessens the nervousness.

The bowels should be properly regulated by cascara, aloin pills, phenolax, etc. Iron springs, such as Franzensbad, or salines, as Kissengen or Wiesbaden, are of service.

The carbonated bath (Nauheim, Triton) I have found—given every other day at home for a course of 12 baths at a temperature of 95° to 98° F.—to be of service in toning up the circulation and general nervous condition.

CHAPTER XVIII

DYSPEPTIC ASTHMA

THIS type of asthma, due to digestive disturbances, was first described by Henoch,[1] later by Silberman,[2] Oppler,[3] Boas,[4] Murdoch,[5] and Einhorn.[6] Many others have described this condition.

The symptoms first reported by Henoch were of an acute type and, according to his belief, were the result of reflex action starting from the stomach. It is noteworthy that the *most severe symptom disappeared when the patient vomited.* In his cases the breathing was rapid and shallow, pulse rapid and feeble, *and at times so rapid* that it could not be counted, extremities cool, the temperature subnormal, and there were even symptoms of collapse.

In all cases there was acute dyspepsia due to some error in diet. The region of the stomach was usually distended and painful. The greatest number of cases were observed in children. Numerous explanations have been given for this condition, and a variety of experiments have been performed. Henoch believes it to be due to reflex action, starting from the stomach and causing a vasomotor spasm; while Einhorn suggests reflex irritation of the vagus fibers. In view of the fact that the majority of cases occur after dietetic error, Riegel's suggestion of auto-intoxication as a factor seems to have a *decided bearing* on the subject.

On the other hand, under acute dilatation of the stomach I referred to certain peculiar clinical types, in one of which many of the symptoms resembled angina pectoris, there being dyspnea and rapid and feeble heart action, and in some attacks loss of consciousness. These attacks followed indiscretion in diet. In view of this fact and also that Henoch describes distention of the stomach as present in most cases, and dietary indiscretion as a cause, it would seem to me that in this type at least acute gastric dilatation from auto-intoxication is a factor. Einhorn describes acute cases following excesses in eating, drinking or smoking, excitement, or from no discoverable cause. Even these facts do not mitigate distention.

The second group which he classifies are more of a chronic type, appearing after meals, or after overexertion, or without apparent cause, or those appearing several hours after meals spontaneously, or after exertion.

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[1] Berlin. klin. Wochenschr., 1876, No. 181.
[2] Ibid., 1882, No. 23.
[3] Allgem. Med. Central. Zeit., 1890, No. 71, p. 849.
[4] Arch. f. Verdauungskrank., Bd. 11, 1896, p. 444.
[5] New York Med. Journal, Jan. 12, 1901.
[6] Jour. Am. Med. Assoc., Feb. 1, 1902.

In some of these cases a small amount of food would check the attack. The last type would seem to suggest reflex irritation from hyperchlorhydria, for example, which when relieved would stop the asthma.

Some of the last group present, in some cases, symptoms suggestive of pseudo-angina pectoris.

Secretory Functions.—Achylia gastrica has been found in some patients and hyperchlorhydria in others. Treatment of these conditions caused subsequent disappearance of the attacks of asthma. In achylia the coarse particles of food, Einhorn believes, might cause reflex irritation of the vagus. Asthma dyspepticum may also occur with chronic gastritis.

On the other hand, these attacks have occurred in patients in whom the gastric secretion was normal; and Boas believes that hyperesthesia of the stomach with reflex irritation is the cause.

Again, in my case of acute dilatation with some of the attacks similar to angina, the gastric secretion was normal; but dietary indiscretion produced gastric distention and the attack noted. It was immediately relieved by vomiting.

Floating liver was noted by Einhorn in 5 cases, and this together with abdominal ptosis of other organs; and he believes that the prolapsed liver, by dragging down the diaphragm, may be a cause of this type of asthma. In my own opinion ptoses of the viscera have an influence on the secretory conditions in the gastro-intestinal tract, and only to this degree predispose to dyspeptic asthma. Probably these various factors to which I have referred may have a bearing in different cases.

Treatment.—Disorders of the gastric secretion must be appropriately corrected, and ptosis of the viscera supported by Rose's adhesive belt.

Excesses in the use of alcohol and tobacco must be corrected, mental worry and strain be avoided, and the mode of life must be regulated.

All indigestible food must be avoided and diet suitable to each case must be instituted. By this means many cases will be relieved and often cured.

In acute cases with distention of the stomach lavage is indicated, also calomel, gr. 5 (0.3), followed by a saline cathartic. Catharsis is also indicated in cases following excesses in eating or drinking.

CHAPTER XIX

THE STOMACH FUNCTIONS IN DISEASES OF OTHER ORGANS

UNQUESTIONABLY there are few diseases, either constitutional or local, which are not attended to a greater or lesser degree by some disturbance of the digestive organs. These are dependent on the general disturbance of the organism and are appropriately discussed under the symptoms of each disease.

In the present chapter I shall only briefly refer to those diseases in which disturbances of the gastric functions are particularly conspicuous.

FUNCTIONS OF THE STOMACH IN ACUTE FEBRILE DISEASES

Numerous investigations have been carried out both on animals and men, in some of which at least accurate quantitative gastric analyses were performed. Riegel concludes that we are probably justified in stating that in acute febrile infectious diseases the production of hydrochloric acid was more frequently reduced than normal, and the secretion of pepsin is, as a rule, unchanged. Probably the fever is responsible and the condition is temporary. Von Noorden[1] showed that hydrochloric acid reaction can be obtained in fever cases if pepper and salt are administered with the food.

Some interesting researches have been carried out in numerous cases of typhoid and pneumonia. During the high temperature of these diseases there was a marked diminution, and in some cases an absence of hydrochloric acid. During defervescence an increase of the secretion was noted. During high tempratures pure milk was found in the stomach in a curdled condition several hours after the normal time for evacuation, and in one case, on autopsy, the stomach was found filled with the curdled milk of previous feedings, thus demonstrating a *diminution of the motor function.* Water alone was then administered at this period. During lower temperatures foods freely soluble in water, such as broths and gruels, were found best. There was less fermentation and distention under this method of feeding and the stomach more readily emptied itself.

In my own experience[2] I can confirm the fact that during the high temperature of typhoid the free hydrochloric acid diminished, often markedly, and the *motor function is not as active.* It has also been found that biliary secretion is disturbed during high fever. Stolnkow noted disturbances in the pancreatic secretion during high temperature.

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[1] Lehrbuch der Pathologie des Stoffwechsels, 1893.
[2] American Medicine, May, 1909.

CHRONIC FEBRILE CONDITIONS

Observers vary considerably in their findings in the gastric secretion in these cases, some noting no changes whatever. In my own experience the temperature, as a rule, caused disturbance in the secretory function of the stomach, lessening the HCl production. The type of disease, the physical condition, the personal equation of the patient, and, most important, *the time at which the analysis is made, all have a bearing.* I shall refer shortly to the work of Hildebrandt on this subject, under Tuberculosis. The power of absorption seems to be impaired in fever (Sticker).[1]

CONDITION OF THE STOMACH IN PULMONARY TUBERCULOSIS

Phthisis quite frequently begins with dietetic disturbances, such as heart-burn, belching, pressure, nausea, loss of appetite, constipation alternating with diarrhea, and even vomiting, with the lung symptoms so slight as to be at first overlooked. It has been called pretubercular dyspepsia. In the later stages we may have the dyspeptic symptoms quite marked.

Hildebrandt[2] found the following results: The cases in which free *hydrochloric acid* was present usually had no fever, while those in whom it was absent suffered from continuous fever. When it was absent at one part of the day, it was when the temperature was high; and when present at another part, it was when the temperature was low. The *temperature, therefore, exercised an influence.* These findings were in advanced cases. Klemperer[3] studied 10 cases in the initial stage, 3 in the advanced, and 1 in the moderate.

In the beginning the secretory power of the stomach was usually increased, often normal, and rarely reduced. In the terminal stages, always greatly reduced. Motor reduction slight in the initial stages, reduced in later stages.

Brieger[4] analyzed 64 cases: 31 advanced, with continuous fever; 37 moderate, with more or less fever; 6 incipient cases, with no fever. Gastric secretion normal in 16 per cent. of advanced cases, and in the others insufficiency of varying degrees; in 9.6 per cent. absence of free hydrochloric acid. In moderate cases, 33⅓ per cent. normal, and in all others perversion; and in 6.6 per cent. normal secretion absent. In the initial cases, normal secretion and perversion were equally divided.

Einhorn[5] has shown in analysis of 15 cases that the appetite does not seem to correspond, as would be expected, to the gastric findings; and also that frequently the subjective symptoms do not harmonize with the objective findings.

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[1] Deutsch. med. Wochenschr., 1889, No. 15.
[2] Berlin. klin. Wochenschr., 1885.
[3] Ibid., 1889, No. 2.
[4] Deutsch. med. Wochenschr., 1889, No. 14.
[5] Loc. cit.

Treatment.—The main indication is to improve the resisting power of the patient against the primary disease.

Forced feeding, especially by Russell's method, rest in bed, and fresh air, with milk and vegetable juices, I believe the best treatment for the tuberculosis. I have seen a gain of 15 to 25 pounds in each case by his methods at Ward's Island in 12 cases in eight weeks.

The heart and kidneys must functionate properly for success with his method. *Functional disturbances of the stomach* should be treated. Tuberculous ulcer of the stomach is occasionally met with in connection with tuberculosis of the other organs.

CHLOROSIS AND ANEMIA

Among the gastric symptoms in these conditions are found gastralgia, anorexia, hyperesthesia of the stomach, hyperchlorhydria, and chronic atony.

These symptoms appear more frequently after eating than on an empty stomach, and occur, as a rule, in attacks at irregular intervals. There are often perversions of appetite. The atony, if neglected, may progress to chronic atonic ectasia.

In chlorosis (primary anemia) the hydrochloric acid secretion is, as a rule, *increased*.

In anemia (secondary), on the other hand, depending on the causative disease we may find variable results; hydrochloric acid decreased, normal, or (more rarely) increased.

In this I agree with Riegel.

The relation between achylia gastrica and pernicious anemia has already been described. The relation of intestinal putrefaction to this disease is described later.

Many of the derangements belong to the neuroses and are dependent on the condition of the blood. The administration of iron is *chiefly indicated*, with the additional correction of the functional disturbance if such be present.

HEART LESIONS

In general we may say that while compensation is present, in many cases the stomach functions are normal or nearly so; with imperfect or failing compensation, with resulting stasis and hyperemia in the gastric mucous membrane, I have noted diminution in the amount of free hydrochloric acid with accompanying digestive disturbances, belching, pressure, anorexia, and at times nausea and even vomiting, with sick headache. These conditions improved after *treatment was directed to the circulation*. In severe cases free HCl may be absent. In one case with poor compensation and frequent gastric attacks, a course of treatment at Nauheim produced excellent results.

Symptoms simulating heart lesions may be produced by gastric disorders, thus: Ulcer, chronic ectasy, and chronic gastritis may produce bradycardia or arhythmia; or tachycardia may occur with chronic gastritis, in nervous gastric disorders, or with atony.

Tachycardia with acute dilatation of the stomach, especially with existing heart lesions, the author has described in the chapter on Acute Ectasy.

DISEASES OF THE LIVER

Diseases of the liver are generally accompanied by gastric symptoms; with cirrhosis and the resulting circulatory disturbances of the viscera the gastric secretion (free hydrochloric acid) is more frequently diminished. The findings in other liver disturbances are not constant.

ANEURYSM

In a case of aneurysm of the celiac axis, referred to under Cancer, free hydrochloric acid was absent, lactic acid present, and the stomach dilated. Circulatory disturbances were responsible for the gastric findings and pressure for the dilatation of the stomach.

DISEASES OF THE KIDNEYS

Gastric disturbances are frequent in nephritis, and nausea and vomiting may be the first symptoms. In fact, Osler has reported death with these symptoms, and nephritis may be unsuspected until autopsy. The excretion of urea through the gastric mucous membrane or cerebral irritation from the poison are responsible for the vomiting. Variable conditions of the gastric secretion have been reported by various observers.

Biernaki[1] has studied 25 cases of nephritis, both acute and interstitial, and found in general the gastric secretion was diminished, and also that the quantity of free hydrochloric acid was reduced in proportion to the extent of the edema, the excretion of albumin, and the reduction in the quantity of urine excreted. Pepsin was reduced and the motor function was increased. Free hydrochloric acid was present in the mild cases in large or small quantities.

Einhorn has observed achylia gastrica in a case of renal calculus, which disappeared after operation, and A. A. Jones[2] has found achylia gastrica among patients with kidney disturbances.

DIABETES

Variable results have been secured. Atrophy of the gastric mucosa has been found in a few cases. The motor power was good.

Rosenstein[3] reports normal secretion in 4 cases and abnormal in 6, while Gans[4] found 6 normal and 4 negative. The findings are not constant, and both normal and abnormal conditions of the secretion have been observed.

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[1] Berlin. klin. Wochenschr., 1891, Nos. 25 and 26.

[2] New York Medical Journal, January 19, 1895.

[3] Berlin. klin. Wochenschr., 1890, No. 15.

[4] IX Congress f. innere Medicin, 1890, Wiesbaden.

ARTHRITIS DEFORMANS

In 1 case[1] I found hyperchlorhydria marked; and Einhorn reports 1 case of achylia.

GOUT

In 2 cases Einhorn reports achylia, and in several mild cases hyperchlorhydria.

Grip.—Gastric disturbances are reported by Kaufmann in grip.

MALARIA

Gastralgia may occur as a substitute for the malarial paroxysms and has already been described, or vomiting may be present, associated with malarial symptoms. There are no characteristic features of the gastric secretion, but in the latter cases free hydrochloric acid may be diminished.

Arteriosclerosis.—General arteriosclerosis may affect the gastric vessels and produce disturbances. Harlow Brooks refers to arteriosclerosis occurring chiefly in the abdominal vessels. The possibility of this latter condition should be considered (see Visceral Arteriosclerosis).

DISEASES OF THE SKIN

Eczema.—Various systemic conditions probably have a bearing, and in some few cases correction of the digestive disturbances seem to have an influence in improving the condition. In one case I found hyperchlorhydria, and in another deficient hydrochloric acid.

Hyde[2] believes that gout, dyspepsia, constipation, and scrofula have a decided influence.

Acne Simplex and Acne Rosacea.—These are associated at times with gastric disturbances. Einhorn reports 2 cases of acne rosacea in whom chronic continuous gastrosuccorrhea was found. The correction of the latter benefited the skin affection.

Psoriasis.—The treatment of gastric disorders in this affection does not seem to benefit the lesion, according to Einhorn.

Urticaria and Erythema.—Some persons have an idiosyncrasy to lobsters, crabs, strawberries, etc., and develop therefrom poisonous substances which produce these eruptions, associated at times with acute gastric symptoms. These conditions seem to be due to auto-intoxication. Combe ("Intestinal Auto-intoxication") believes that acne, the seborrheic eczemas, urticaria, pruritus, strophulus infantum, and furunculosis to be chiefly due to intestinal auto-intoxication. He advises fresh brewers' yeast, ʒj (4.0) t. i. d., before meals for these conditions. Duncan Bulkley believes that cutaneous lesions are in some cases produced through cutaneous elimination of toxic substances. The author holds that the entire gastro-intestinal tract should receive attention. Thorough catharsis should be carried out. These foods should thereafter be avoided.

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[1] The relation of intestinal putrefaction to arthritis deformans is described later.

[2] Twentieth Century Practice, vol. v.

Pemphigus of the Mouth.—Einhorn has noted 3 cases in which there was hyperchlorhydria or neurasthenia gastrica, and in 2 cases improvement resulted from treating the gastric symptoms.

In general, we may say that considerable investigation is still necessary to definitely determine the relations of gastric disturbances to skin diseases.

SYPHILIS OF THE STOMACH

Gastric symptoms quite frequently occur in the secondary and tertiary stages of syphilis. In the secondary stage they may often be attributed to fever (the constitutional condition); while in the tertiary stage there are anatomic changes in the stomach itself.

Fenwick[1] believes that syphilis may affect the stomach in three ways: By the formation of gummata, by producing endarteritis, and by a chronic inflammation of the mucous membrane. The symptoms arising subside under antisyphilitic treatment.

Flexner[2] holds that syphilitic gastric ulcer is not rare, while Dieulafoy[3] notes various lesions, such as hemorrhagic erosions, ecchymoses, gummata, infiltration of the submucosa and circumscribed gummatous ulceration, and cicatrices of the latter. Pain, vomiting, hematemesis, and melena occur.

Riegel[4] reports 12 cases in which they complained of gas, nausea, distress after eating and gastralgia, which responded promptly to antisyphilitic remedies.

Death has resulted from perforation of *a broken-down gumma.*

All doubtful cases should be examined for signs of previous syphilitic infection,[5] for active syphilitic manifestation; also as to the history, and should be tested by specific treatment. Of course, many patients may have digestive disturbances without any connection with the old luetic condition.

Hemmeter[6] has described syphilis of the stomach.

Einhorn[7] gives the following classification and describes cases:

(1) Gastric ulcer of syphilitic origin.

(2) Syphilitic tumor of the stomach.

(3) Syphilitic stenosis of the pylorus.

To this I shall add a *fourth type:*

(4) Syphilitic cirrhosis of the stomach.

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[1] London Lancet, Sept. 20, 1901.

[2] Am. Jour. Med. Sciences, Oct., 1898.

[3] Gaz. heb. de Med., 1902.

[4] Diseases of the Stomach.

[5] The test for Wassermann's reaction is important, both as confirmatory and for the diagnosis of doubtful cases.

[6] Diseases of the Stomach, p. 556, 1897.

[7] Ibid., p. 534, 1906.

(1) Gastric Ulcer (Syphilitic)

A number of cases have been reported in which the usual treatment for ulcer failed, and which made complete recovery under specific treatment.

(2) Syphilitic Tumor of the Stomach

This condition is excessively rare. Einhorn has reported 2 cases, and refers to the fact that they may run their course like carcinoma. I referred to a case in this volume under Differential Diagnosis in Carcinoma of the Stomach. In this patient the gastric analysis showed absence of hydrochloric acid and lactic acid abundant. The patient had lost 77 pounds in eight months and was vomiting continuously. The stomach was dilated to below the umbilicus, and though he had been on specific treatment for a time before I saw him, the pyloric obstruction was so marked that drainage of the stomach was necessary to preserve life. Palpation gave a sense of resistance at pylorus.

A rapid laparotomy at the Red Cross Hospital disclosed a gummatous tumor at the posterior wall of the pylorus, nearly blocking it. Gastro-enterostomy was performed and specific treatment pushed. There was no more vomiting and the case has steadily improved.

(3) Syphilitic Pyloric Stenosis

Einhorn reports a case of pyloric stenosis cured by antisyphilitic treatment. In most of these cases, however, gastro-enterostomy is required in addition to the antiluetic treatment.

(4) Syphilitic Cirrhosis of the Stomach

This case has also been referred to under Cancer. The stomach was small, hard, and contracted, and on palpation felt like a cirrhotic carcinoma, involving the entire stomach. The patient was an elderly man, had lost considerable weight, and was suffering from gastric symptoms.

Examination demonstrated cirrhosis of the liver and evidences of old syphilis. Deficient HCl, or achylia, occurs in this type.

These facts show that the syphilitic history, or evidences of the same, should be investigated carefully in gastric affections.

Treatment should be for syphilis. Appropriate remedies may be given in addition for special symptoms, or secretory or motor disturbances.

PART III

DISEASES OF THE INTESTINES

CHAPTER XX

METHODS OF EXAMINATION OF THE INTESTINES; EXAMINATION OF THE FECES; MECHANICAL PROCEDURES.

Special Interrogation.—We presuppose that the method of interrogation of the patient, as described in Part I, has been carried out. Disease of the stomach may *produce secondary intestinal symptoms*, and hence the condition of this organ must be inquired into, and in many cases the functions examined.

Inquire, furthermore, whether abdominal pains are present, and also their position. In the right iliac fossa they suggest appendicitis or catarrh of the cecum; in the left iliac fossa, disturbances of the descending colon or sigmoid.

With rectal pain, inflammation in that region is probable, while pains near the navel usually originate in the small intestine. Pains of short duration and sharp in character are generally due to colic, and are followed and relieved by the passage of flatus or feces. They often shift from one region to another. Pains of long duration are usually from some *organic lesion*, such as ulcer or from some affection of the sensory nerves.

Abnormal sensations, such as feelings of heat or cold, may be experienced over different regions of the abdomen.

Tenesmus is present in dysentery and in many rectal affections. The time of the pain, whether immediately after meals or later, or during the night or in the early morning, is important.

The stool should be investigated—whether constipation, diarrhea, or alternating conditions exist, the number of movements, time of appearance, odor, color, and characteristics, as to whether mucus, blood, bile, pus, or undigested food are present.

Do climatic changes or mental excitement influence the bowel action, or are headache, dizziness, or exhaustion associated with the movements? Is there distention of the abdomen with gas, localized or general? When does it appear? Is rumbling (are borborygmi) present? Do belching of wind or passage of gas from the bowel occur, and does this give relief?

Total absence of flatus is important. Occurring with obstinate constipation, it would then suggest obstruction.

Continuous vomiting associated with intestinal symptoms suggests obstruction. With acute symptoms the temperature should be taken immediately.

PHYSICAL EXAMINATION OF THE INTESTINES

The reader is referred to Chapter IV for general methods.

Inspection.—The retracted or trough-shaped abdomen occurs in stricture of the esophagus or cardia, basilar meningitis, lead-poisoning, and with long-continued inanition. The peculiar contour of enteroptosis has been described—the concave epigastrium, sulcus between the recti above the umbilicus, and the pot-belly below.

On distention of the normal colon with gas (CO_2), the ascending and descending portions are seen as elongated swellings in the lateral regions, and the transverse colon at or just above the umbilicus.

Protrusion of the abdomen may be over a definite area or over the entire surface. It may assume the shape of a rounded hemisphere, or oval, slightly flattened, especially in atonic conditions of the intestines and in hysteria. Marked uniform distention with tense abdominal walls, *absence* of respiratory abdominal movements and increased thoracic respiration are present in peritonitis. There may be a general bloating with atony, but there is not the marked tension of the abdominal walls, and the other symptoms are absent.

With ascites the abdomen is evenly protuberant above, with the center somewhat flattened, while the lateral and dependent parts bulge *somewhat* in the recumbent position; change of posture alters the shape of the abdomen. This applies to the milder types.

With marked distention, as with meteorism, the enlargement is uniform. There is no bulging in any special location, except that the anterior portion is more prominent and change of position has no effect. Palpation aids under these conditions.

There may be protrusion of the abdomen in cases of neoplasm, in fecal accumulation, and occasionally in abscess, as of the appendix, from diverticulitis or other intra-abdominal suppuration.

Hernial protrusions at the umbilicus or in the inguinal regions may be observed.

In patients with thin abdominal walls, small sausage-shaped protrusions are occasionally visible, which change their shape and position. This is due to peristalsis of the small intestine, occurs with no pain, and denotes no morbid condition.

Similar waves may appear periodically and annoy the patient when caused by nervous influences.

There are sometimes violent contractions (peristaltic unrest) of the small intestine visible, caused by stenosis or obstruction. If it is near the ileocecal valve, the swollen and moving coils of intestine lie one above the other in the central part of the abdomen (ladder pattern). Intense pain accompanies these movements.

Marked distention may be visible in the course of the colon (in the circumference of the abdomen), and if associated with visible peristaltic contractions of the large intestine, passing along it from right to left, it is diagnostic of partial or total obstruction of the large bowel.

In some cases a recurring protuberance is noted, disappearing with a loud sound. This is probably near the point of stenosis.

INSPECTION OF THE RECTUM—PROCTOSCOPY AND SIGMOIDOSCOPY

The anus can be inspected by having the patient lie on his side with thighs and knees flexed, and his back toward the examiner.

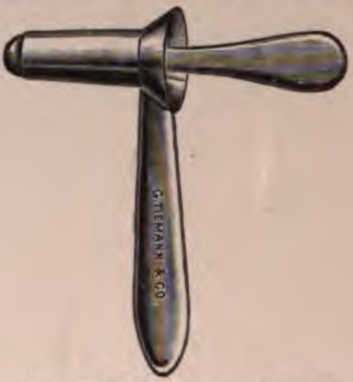

Fig. 162.—Kelly's short rectal speculum.

The buttocks should be held apart with the hands. Hemorrhoids, fissures, and fistulæ may be discovered.

For inspection of the rectum the introduction of a speculum (proctoscope) is necessary.

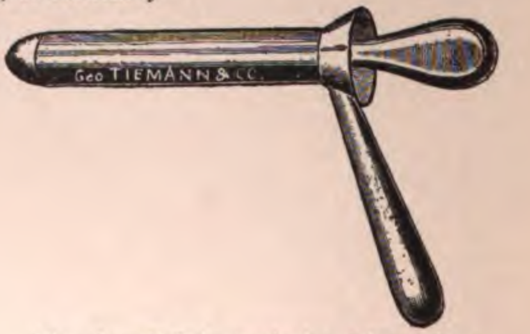

Fig. 163.—Kelly's standard rectal speculum.

Proctoscopy.—Various instruments have been devised, notably those of Howard Kelly, Sims, Kelsey, Gant, and J. P. Tuttle (Figs. 162–170).

The bowels should preferably be thoroughly evacuated before the examination. If the region is sensitive, a few drops of a 2 to 5 per cent. cocain solution can be injected inside and along the sphincters with a narrow-pointed rubber syringe. A suppository containing opium, gr. j (0.065), with extract of belladonna, gr. ⅓ (0.022), or cocain, gr. ⅛ (0.008), can be substituted.

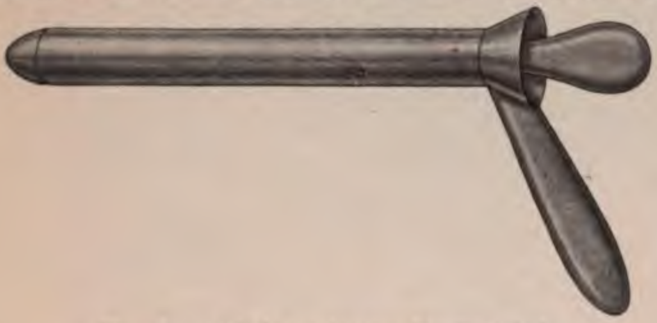

Fig. 164.—Kelly's proctoscope (⅓ actual size).

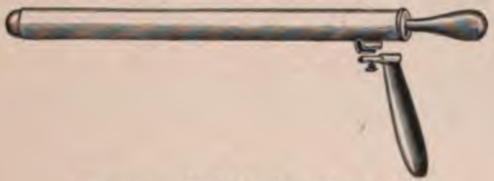

Fig. 165.—Kelly's sigmoid speculum.

Tuttle's pneumatic proctoscope is a valuable instrument. There is an electric lamp at the end of the inspection tube and an arrangement for inflation of the rectum, so that it can be distended with air at the time of examination.

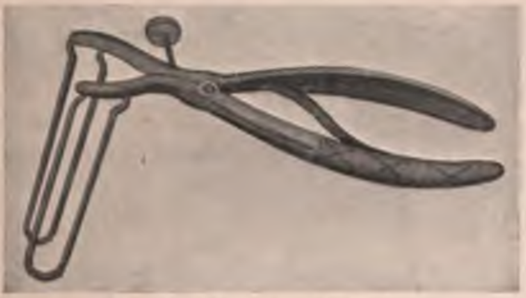

Fig. 166.—Sims' rectal speculum.

With other specula, a head-mirror with electric-light attachment is most convenient, though an ordinary light can be arranged. The patient lies on the side, with thighs and knees flexed, with back toward examiner, or the legs can be elevated on a crutch, or in some cases the knee-elbow position can be assumed. The external sphincter as well as the speculum should be lubricated with sweet oil or vaselin, to render introduction more easy.

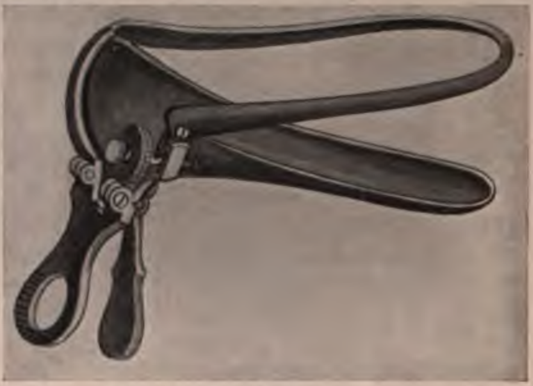

Fig. 167.—Kelsey's speculum.

A long speculum (sigmoidoscope) may be required for examination of the sigmoid.

Palpation.—The technic of simple and reinforced palpation has been described in Chapter IV.

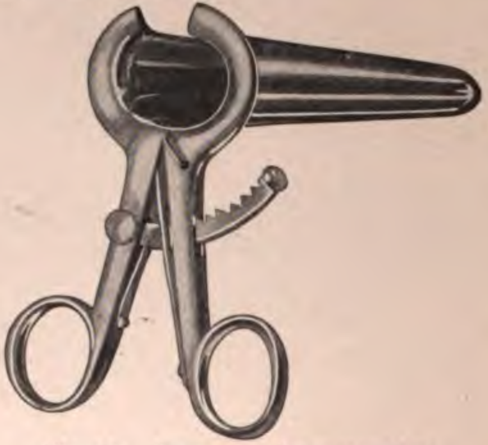

Fig. 168.—Gant's examining speculum.

The cecum, parts of the ascending and descending colon, the transverse colon, and the sigmoid flexure are often accessible to palpation, but not as readily so in obese or in muscular subjects. Fecal accumulation, tumors, thickening of the gut, or purulent collections connected with the intestine can often thus be recognized.

An uneven protuberant surface is characteristic of malignant growth, while an even surface is more often found in benignant neoplasm or intussusception. Volvulus occurs usually in the sigmoid. A fecal accumulation will, as a rule, "pit" on pressure (give a doughy feel). Hard scybalæ occasionally feel like marbles under the fingers, but can be moved or slightly indented. Sometimes when raising the fingers from palpating, there may be a crepitating or

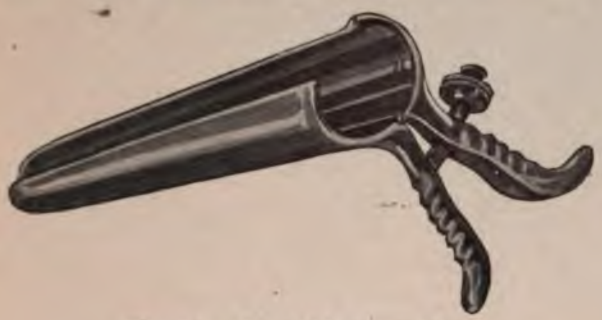

Fig. 169.—Gant's hinged speculum.

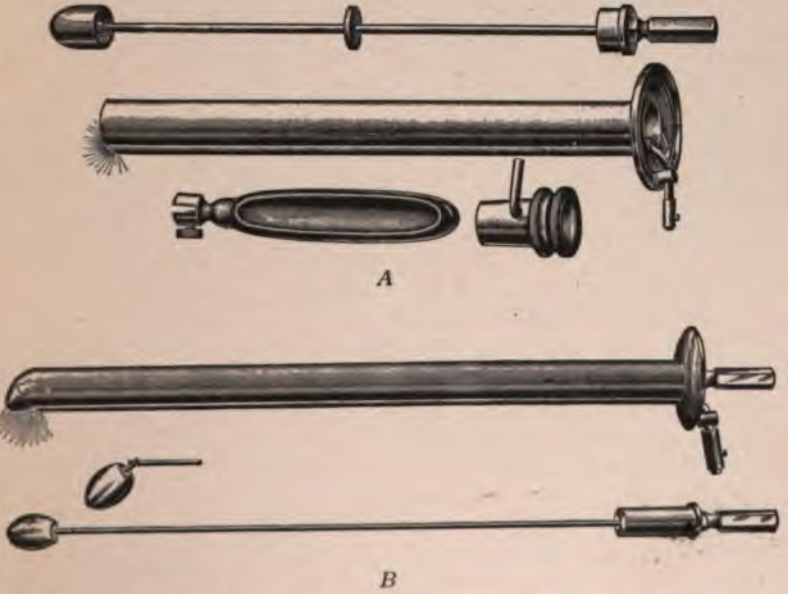

Fig. 170.—*A*, Tuttle's pneumatic proctosigmoidoscope. Two lengths—rectal 4 inches, sigmoid 10 inches—with window attachment to make instrument air-tight for bowel inflation; *B*, Tuttle's sigmoidoscope with Mercier curve.

sticky sensation, or the intestinal wall can be felt to slip off from the fecal mass. This symptom was first described by Gersuny.[1]

Gurgling occurs on palpation in typhoid in the right iliac fossa, but is not diagnostic.

Tenderness or pain on pressure can be readily determined by palpation, and are suggestive of inflammatory processes or ulceration. There may be the general tenderness of acute intestinal inflammation or the acute pain and tenderness of peritonitis, either localized or general.

Circumscribed pain on pressure may be present at McBurney's point (1½ to 2 inches to the inner side of the anterior superior spine of the right ileum) on a line drawn from this process to the umbilicus.

With ulceration of the bowels there may be circumscribed areas very sensitive to pressure; with hysteric manifestations there are often sensitive spots complained of in the abdomen, as for example, in mucous colic. By palpating simultaneously two distinct points, the supposed painful area and another region, with the different hands and at the same time distracting the patient's attention by conversation, one often finds an absence of true tenderness at the supposed seat of pain.

Muscular rigidity shows peritonitic involvement. It may be localized, as of the right rectus in the region of the appendix or gall-bladder, or over the left rectus, as in diverticulitis or phlegmonous gastritis, or in abscess of the left lobe of the liver. General rigidity shows general peritonitis.

Splashing Sound (Clapotage, Succussion).—If the intestines contain liquid material and gas, tapping over them with the fingers will at times produce the splashing sound. The method of differentiation between stomach and intestinal splash has been described under Splash of the Stomach in Chapter V.

In the small intestine clapotage can usually only be obtained in the dilated portion of the gut above a stricture. It is not uncommon in the large intestine, and can be most often determined in the sigmoid flexure, caput coli, and the transverse colon. In case of atony of the bowel it is quite frequently present, also in the relaxed abdomen of enteroptosis, and often in patients with hysteric manifestations.

Boas[2] first suggested injecting into the bowel 500 cc. (1 pint) or more of water and then examining for the splash along the colon. It should be given with *hips elevated*. The splash will first be secured in the sigmoid, and by turning the patient on the right side it can at times be produced in the transverse colon and in the cecal region.

It is possible to administer an enema of moderate size and cause it to gravitate to the caput coli by the method of rotation described under Enteroclysis. By the splash one can determine whether an external sphincter, so that the distance of the stricture up the bowel can be estimated. Smaller tubes are then employed until one can pass the obstruction. Its caliber is thus estimated. The ordinary soft flexible rectal or colon-tube is the safest for diagnostic purposes in the hands of the general practitioner.

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[1] Wiener klin. Wochenschr., 1891, No. 4.

[2] Diagnostik und Therapie der Magenkrankheiten, 1897.

In Fig. 171, *A* and *B*, are shown an olive-pointed flexible and a cylindric bougie. The latter is somewhat stiff and can be softened in hot or boiling water before use. This last is also employed for dilatation of the stricture.

Kuhn's metal spiral tube is of no advantage. Care should be exercised if stiff tubes are employed.

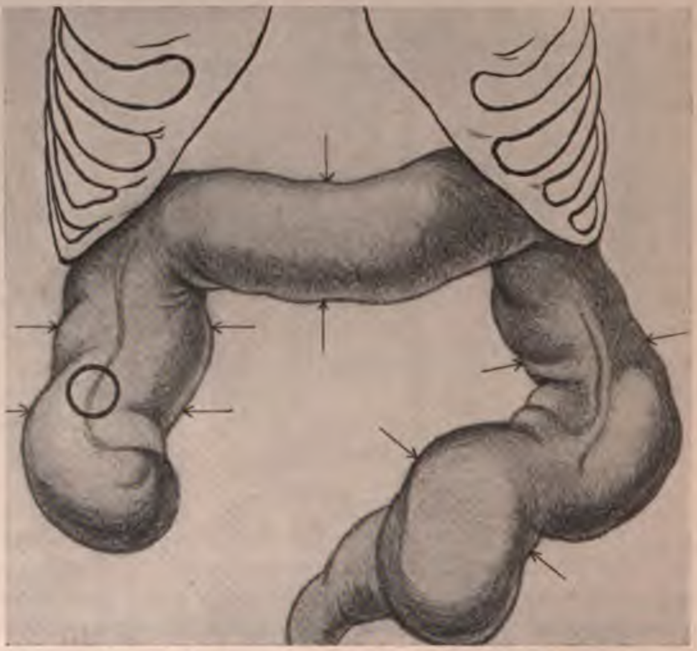

Fig. 172.—Auscultatory percussion of the colon.

Percussion should be gentle. Over empty intestinal coils or those containing gas or air, a tympanitic sound results, which is louder over the large than over the small bowel. As there may be considerable distention of the small intestine, it is sometimes difficult to delimit the large intestine by simple percussion. If the colon is emptied by enema, and then distended artificially with air or carbonic acid gas, the procedure is much easier.

Intestinal coils which are filled with liquid or solid material give dulness on percussion. With meteorism there is tympanites of a deeper pitch than normal, and sometimes there is a metallic sound with auscultatory percussion. The meteorism may be localized or general. If local, in connection with visible peristalsis, intestinal stenosis is at once suggested. With local meteorism there will be dull areas elsewhere; with general meteorism, the entire abdomen is symmetrically distended, the anterior portion being most protruded, and there is the diffused tympanitic note of the peculiar type noted, and dulness over the region of the liver and spleen may disappear. With ascites, percussion shows dulness in the lower lateral regions of the abdomen and tympanites in the middle. The sounds change on altering the position of the patient (turning him on his side). The intestines ride up on the fluid, and the upper flank, previously dull when in the dorsal position, is now tympanitic.

Fecal accumulation, tumors, and abscesses give dulness on percussion.

Auscultatory Percussion.—This is the best method of determining the position of the colon. If the small intestine is excessively distended it is difficult to differentiate the percussion sounds. It may be necessary to empty the colon by enema or irrigation and then inflate with air or CO$_2$. Place the stethoscope at the circle (Fig. 172) over the cecum; begin percussion midway between the umbilicus and symphysis, and percuss to the right, to the left, and upward, in the direction of the arrows, until in each direction the greater intensely altered quality and heightened pitch show that the inner border of the colon has been reached. These points can be marked on the abdomen with a pencil. Then percuss in the epigastric region (midway between the ensiform and umbilicus) downward, and from the lateral lumbar regions inward. The changes in pitch, quality, etc., should be marked, and thus the outer limits of the transverse ascending and descending portions of the colon are determined.

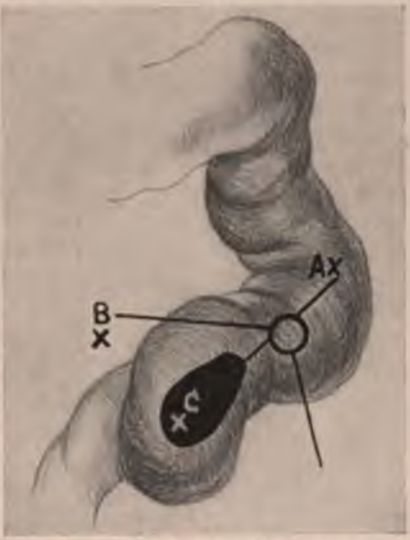

Fig. 173.—Differential percussion.

The scratch method of auscultatory percussion may be carried out in the same lines.

Stengel claims that by auscultatory percussion it is possible to determine that a tumor found to lie in the course of the intestine originates in the wall of the colon. In Fig. 173, C represents the tumor; the circle O, the stethoscope placed over the colon near the tumor; B, percussion note over small intestine; A, percussion over normal large intestine near the stethoscope.

First percuss directly over the tumor, then toward it from every direction. The note over tumor, C, if it is connected with the colon, resembles the percussion note at A (colon) more closely than does percussion note at B (small intestine) resemble note at A (colon).

Auscultation.—This is not of great significance in intestinal diseases. Palpation may elicit a gurgling noise in the right iliac fossa, formerly thought to be pathognomonic of typhoid, but it is found in many other conditions. Gurgling sounds (borborygmi) are at times heard. They may be due to fermentative processes or occur in neuroses, and are not specially significant.

The entire absence of intestinal sounds may be significant of intestinal paresis. If the latter is due to peritonitis, cardiac and respiratory sounds may be audible over the entire abdomen. Friction sounds from perisplenitis or perihepatitis may rarely be auscultated. With chronic stenosis very loud noises are at times heard, caused by the sudden passage of fluid and gas through the stricture under pressure.

Splashing sounds are at times distinguishable in the enlarged bowel above the stricture.

TRANSILLUMINATION OF THE INTESTINES

This method was first suggested by Einhorn[1] and further practised by Heryng and Reichmann.[2]

Einhorn's technic is as follows: A high enema or irrigation of the bowel is first given. A quart of water is later injected per rectum and the illuminator, very similar to the gastrodiaphane, is inserted and gradually pushed up the intestine. The examination must be made in a dark room. Experiments were carried out by the author with specially devised illuminators at the Manhattan State Hospital.

As the average adult rectum is 8 inches long, the sigmoid flexure 17½ inches, and allowance must be made for the sphincters, it requires an instrument *at least 30 inches in length* to pass through the sigmoid into the descending colon. The 30-inch instruments, with which I experimented, on almost every occasion caught and coiled back, and rarely did I succeed in securing transillumination of the lowest part of the sigmoid, and even then the light was so faint that it was entirely unsatisfactory.

Inflation of the bowel with water, with fluorescein solution, and with air were all tried before passage of the light, as were various positions of the patient.

The experiments demonstrated practically the impossibility of passing a flexible instrument or tube through the sigmoid. The sigmoid is quite movable, and Howard Kelly has shown that the colon-tube readily pushes it up. My experiments demonstrated visually the impossibility of passing the long colon-tube through the sigmoid flexure and showed that transillumination of the sigmoid has not been sufficiently certain to prove of practical value.

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[1] New York Med. Monatsschrift, Nov., 1889.

[2] Therapeutische Monatshefte, 1892.

Röntgen Rays; (*x*-Rays).—For examination of the intestines the *x*-rays are of value for the following conditions:

The determination of the presence of a foreign body in the intestinal tract, accurately locating its position, and hence the site for operation.

Einhorn[1] has recommended the internal administration of bismuth subnitrate in watery solution to locate the constriction in acute intestinal obstruction with the aid of the *x*-rays. The delay necessitated would be dangerous.

For locating the seat of chronic intestinal occlusion the method may be of service.

An ounce (30.0) of bismuth subnitrate or bismuth subcarbonate can be administered in ℥xij (375 cc.) of milk or water, and about twenty-four hours later examination should be made with the *x*-rays. With the fluoroscope or by a photograph one would see a distended area of intestine filled with bismuth, a region below with apparently no bismuth or a trace (the point of stenosis), and below this a small amount of bismuth that had passed through the stricture. If the stricture is apparently in the large intestine, a check test can be made as suggested by Einhorn.[2]

A few days later, when the bowel is free from bismuth, an injection per rectum is given of 500 cc. (1 pint) of water containing 30 gm. (about ℥j) of bismuth subnitrate. If the stenosis is in the large intestine it will be located by the Röntgen picture, there being the area apparently free from bismuth (the stricture) and the collections above and below the stricture.

Location of the Colon by the x-Rays.—Two quarts (liters) of water, in which 60 gm. (about 2 ounces) of subnitrate of bismuth are suspended by means of a little starch solution, are injected per rectum, with the hips elevated or in the knee-elbow posture. The position of the colon can be immediately determined by the Röntgen ray.

One could administer the bismuth by mouth and examine at the end of twenty-four hours, but the enema method is preferable. Misplacements of the colon, enteroptosis, and angulations of the sigmoid can thus be determined.

It has been claimed that a soft tube, in which lies a flexible wire, can be introduced per rectum, and the course of the colon determined by the *x*-rays, the wire showing a shadow. It is practically impossible to insert the tube beyond the sigmoid, so the method is not accurate. The *x*-ray pictures shown of the long colon-tube—*supposedly in the descending colon*—are usually in the ampulla of the rectum. H. W. Soper[3] has demonstrated, by means of the *x*-rays that it is impossible to pass the colon-tube into the sigmoid, except in the case of Hirschsprung's disease (congenital idiopathic dilatation and hypertrophy of the colon).

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[1] New York Med. Journal, May 18, 1907.

[2] Ibid.

[3] Jour. Am. Med. Assoc., Aug. 7, 1909.

For *locating the position of the colon* the Röntgen method is expensive and usually impractical, as it gives no more information than by inflation.

Physiologic Investigations with Röntgen Rays.—Cannon[1] has investigated intestinal movements after administration of bismuth by means of the *x*-rays. He experimented on cats, and claims there are periodic antiperistaltic movements in the cecum and ascending and transverse colon which aid in churning and mixing the contents, and so help absorption. He holds that some of the material may be forced back into the small intestine, and that this may occur with a high nutrient enema.

Some investigators disagree with Cannon. Grützner has shown that starch granules, lycopodium, powdered carbon, etc., in physiologic salt solution, injected into the bowel under favorable circumstances, will find their way upward into the stomach.

By means of the *x*-rays Hemmeter observed that the upward movement of these particles goes on simultaneously with the downward movement of the feces, *i. e.*, there is upward marginal current. He considers the epithelia and muscularis mucosa instrumental, and that it is not true antiperistalsis.

INFLATION OF THE INTESTINES WITH CARBONIC ACID GAS OR AIR

Von Ziemssen[2] first employed inflation of the colon for diagnostic purposes by injecting in succession into the bowel solutions of tartaric acid and sodium bicarbonate, with the resulting development of carbonic acid gas. The bowel could then be recognized by the marked tympanitic sounds on percussion or, more rarely, by inspection. Preferably, the gut should be previously emptied by enema.

Inject into the bowel ʒj (4.0) of tartaric acid, which has been dissolved in ℥vj to viij (200–250 cc.) of water, and follow it by the injection of the same quantity of soda bicarbonate in same amount of water. Preferably the hips should be elevated.

Schnetter suggested attaching a flexible tube with rectal tip to the nozzle of an inverted soda-water siphon and driving out the CO_2 by pressing the valve.

The gas has also been injected by obtaining it from the liquefied gas in a sparklet and conducting the CO_2 from a bottle into the rectum.

One of the *simplest methods to inflate the bowel by means of carbonic acid gas* is by Rose's gas bottle. This consists of a moderate-sized bottle with perforated cork, through which passes a glass tube. To this is attached a soft-rubber tube and rectal tip (Fig. 174).

The bottle is half-filled with water and ʒj (4.00) each of tartaric acid and soda bicarbonate added, and the cork tightly inserted. The accumulating gas is conducted off by the tube into the rectum. Inspection and percussion will determine the extent of distention.

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[1] American Journal of Physiol., vol. vi, p. 253.

[2] Deutsches Archiv. f. klin. Medizin, 1883, Bd. 38, S. 325.

Runeberg[1] recommended inflation of the intestines by air by means of a colon-tube, to which a compressible air bulb is attached. It is possible by this means to measure and regulate the quantity of air employed for inflation.

An ordinary Davidson's syringe can be used to pump in the air, and its capacity can be determined as follows:

Take a measuring glass of 1 pint (500 cc.) to 1 quart (liter) or a glass vessel of unknown capacity and measure its capacity when filled to the brim. Invert the filled vessel in a pail of water, so that the entire column of water is sustained in the inverted vessel. Then slip the colon-tube under water, so that its tip enters the inverted jar. Observe how many compressions of the bulb are required to replace the water column with air, that is, to drive out all the water from the inverted vessel.

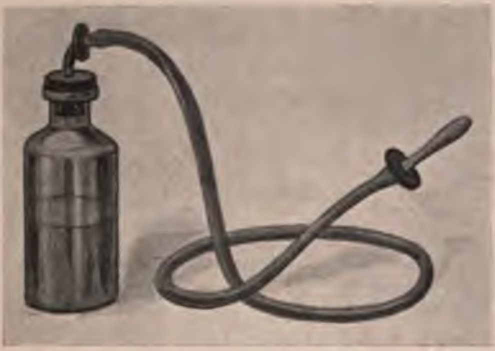

Fig. 174.—Rose's carbonic acid gas generator.

If, for example, the jar contained 500 cc. (1 pint) and it required sixteen squeezes of the bulb to replace this by air, then each squeeze of the bulb replaces 1 ounce of water by the air equivalent.

Uses of Inflation.—It is of service to detect stenosis of the large intestine. Under normal conditions the injected air distends the colon evenly. If there is stenosis, the air will distend chiefly that part of the bowel below the stricture, while above it remains unchanged. This is true in marked strictures, but in those of mild type the air will pass through. Even in some such cases there will be less distention above than below the strictured point.

The *position* of the colon can be determined by air inflation. Normally it passes with a slight downward curve across the abdomen with lower edge about touching the upper margin of the umbilicus.

Enteroptosis is demonstrated by this method and the transverse colon may descend to a hand's breadth above the symphysis. It may assume a **V** shape. Angulations of the sigmoid may at times be determined by this means.

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[1] Deutsches Archiv. f. klin. Med., Bd. 34, S. 460.

For the diagnosis of the location of abdominal tumors inflation of the intestine is often of service. After inflation of the colon with air, tumors of the viscera become more distinct; while tumors of the kidney, retroperitoneal glands, spine, etc., tend to disappear.

Minkowski[1] holds that after filling the colon with air or water, abdominal tumors are shifted in the direction of the organ to which they belong.

Sutton[2] suggests inflating the bowel with air impregnated with ether for the diagnosis of intestinal perforation. He employs a bottle provided with a perforated rubber cork to which are attached two rubber tubes with stop-cocks. To one of these tubes is attached a bicycle pump or a Davidson's syringe; to the other an ordinary colon-tube by means of a short glass connecting tube.

Two drams of ether are placed in the bottle. The air pumped into the bowel passes through the bottle and thus takes up the vapor of ether. With the stop-cocks the pressure of ether and air can be regulated.

If perforation of the bowel is present, the ether escapes through the opening into the abdominal cavity and distends it equally in all directions. If there is no perforation, first the large intestine and later the small intestine become filled with air and ether, and finally ether vapor may be eructated and readily recognized. This method is useful in gunshot wounds of the abdomen.

Inflation of the Colon with Water.—This can be performed with a graduated irrigating jar and a rectal tube. With a fountain syringe of known capacity it is easy to estimate the quantity injected.

In *stricture of the colon*, especially in the lower portion, the quantity of water which can be injected is not great. Normally the colon will contain from 3 to 4 quarts, or occasionally 5, without dangerous distention. The injection should be given with hips elevated. *Many people are unable to hold any quantity of water in the bowels without pain, discomfort, and rapid evacuation of the fluid.*

Determination of the position of the colon by inflating it with water is, therefore, often difficult. The air inflation is preferable for the above reasons.

LAVAGE OF THE BOWEL FOR DIAGNOSIS

Boas[3] first recommended this procedure to be carried out in a manner similar to lavage of the stomach. The bowels should be previously evacuated. The patient should lie on the side with the knees and thighs flexed. The colon-tube is attached by a short piece of metal or glass tubing to a long tube, provided with a funnel. The rectal tube is lubricated and inserted high up to its full length and about 500 cc. (1 pint) to 1 liter (quart) gradually poured in through the funnel held 1 or 2 feet above the patient, until some discomfort is expressed. The funnel is then lowered below the anus and the contents siphoned off.

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[1] Berlin. klin. Wochenschr., 1888, No. 31.

[2] Journal Am. Med. Association, Dec. 30, 1899.

[3] Deutsch. Aertze-Zeitung, 1895, Nos. 2 and 3.

They are then submitted to a thorough examination. Normally the contents are fairly clear or slightly stained with fecal matter and contain a little mucus and a few epithelial cells.

With intestinal catarrh a large amount of mucus is present. Blood or pus may be found, showing hemorrhages or a suppurative process, such as an ulcer or abscess. Exfoliated portions of the mucous membrane may occasionally be found or, rarely, tumor fragments.

Microscopic examination of such material is of diagnostic importance. Intestinal worms are occasionally discovered. Lavage of the bowel is also employed for the removal of dysenteric discharge and mucus, for the purpose of examination for amebæ.

EXAMINATION OF THE FECES

The Stool.—*General Considerations.*—The normal stool consists of changed and unchanged remnants of food, bacteria (estimated at about 126 billions daily), epithelial cells, salts, and traces of the digestive juices.

The normal daily quantity under a mixed diet averages 100 to even 200 gm. (3½ to 7 ounces). It may be increased by a vegetable diet. There is usually one movement daily of dark brown color, though diet and medicine have an influence. Milk gives a light yellow; claret and huckleberries, a brownish black; salts of iron and magnesia, a blackish brown; bismuth, black. Blue is given by iodids (long continued); green, by calomel; yellow, by santonin, senna, and rhubarb; violet, by salol and betanaphtol. The feces are slightly soft and of sausage shape. Abnormally they may appear in small balls, cylinders or tape-like, or as hard scybalæ (dry in character), or they may be mushy or liquid. They may be very watery, as in choleraic conditions, or fluid and mixed with mucus.

Odor.—This is normally caused by skatol and slightly by indol. It is increased when the feces have been retained an abnormal time. After a short sojourn in the intestines, as with rice-water movements, there is often no odor. The character of the food may affect the odor. Very fetid movements occur with ulcerative processes or with malignant growths.

Macroscopic Findings.—*Remnants of Food in the Feces.*—Undigested remnants of food can often be seen in the stool. Normally they consist of only small particles of vegetable material, such as potato, asparagus, spinach, and peas; while *remnants of meat* cannot be seen. Providing abnormal quantities of food have not been ingested, it is often possible to draw definite conclusions as to the state of intestinal digestion, from the excess of one form of non-digested material over another. The presence of *large quantities of undigested starch indicates a catarrhal condition of the small intestine*, and, indeed, more *than traces* of this material should be *regarded with suspicion.* If particles of meat are visible, this indicates a lesion of the intestinal tract. *Connective-tissue fibers* appearing unaltered in the feces indicate deficient gastric digestion, according to Schmidt, and the presence of nuclei, under the microscope, disturbance of the trypsin function of the pancreas.

Blood.—Blood may be visible in the feces, either fresh (red) or dark in color and uncoagulated, which shows its origin from the lower part of the large bowel. It may appear changed, giving the feces the appearance of tar, then originating from the small intestine or even from the stomach. Blood shows the presence of an ulceration or of an ulcerating cavity communicating with the gut.

Pus.—Visible pus in the dejecta only occurs when pus exists in large quantities in the lower part of the large intestine. It shows ulceration or an abscess communicating with the gut. Pus in small quantity or from higher up the intestine can only be determined by the microscope.

Fragments of tumor (polypi or cancer) may rarely be found in the dejecta. Microscopic examination will give important information.

Mucus.—Mucin can always be detected in normal feces by chemic examination. The amount of mucus in the feces in health is so small and so intimately mixed as to be only recognized by chemic tests. Mucus in the stool, either macroscopic or microscopic, indicates some deviation from *the normal physiologic condition.* It does not *invariably show an anatomic lesion.* We may, for example, have a few flakes of mucus or an extremely thin layer adherent to scybalæ, due to irritation of the mucosa from a fecal accumulation or impaction, or mucus, which is contained normally in the higher portions of the small intestine, may occasionally appear in the stool as a result of increased peristalsis.

Under other circumstances the presence of mucus is pathologic. Macroscopically, mucus may exist as follows:

(1) An abundant coating in the form of a glassy layer may cover fecal masses. It may be gray or cloudy from epithelial or round cells. This usually indicates catarrh of the lower portion of the bowel.

(2) It may be intimately mixed with the feces in mushy movements, and may adhere to a glass rod if this is dipped in the stool.

(3) It may float on top of watery movements.

(4) It may be passed almost pure in large amount.

Material resembling *frogs' spawn* or sago grains may occur in the feces. They were formerly considered due to follicular ulceration, but are now believed to be of vegetable origin. Kitagawa holds that some of them are pure mucus, but that they are not pathologic.

Yellow or yellowish brown granules occur in the stool, from the size of a pinhead to a poppy seed, resembling butter in consistency. Some of these have been considered bile-stained mucus, while other fragments are believed to be albuminous or vegetable material or yellow calcium salts.

Boas and Schmidt believe these yellow granules to be albuminous matter stained with bile-pigment.

The presence of any one of the previously described types of mucus indicates intestinal catarrh. Mucus without feces or surrounding the feces shows the colon is inflamed. When mucus is mixed with the feces the upper colon or small intestine is inflamed. Mucus in the food residue shows catarrh of the small intestine.

There are two exceptions to the rule that *visible mucus indicates catarrh:*

(1) In mucous colic (membranous enteritis) pure mucus due to hypersecretion is evacuated in the form of a cast membrane or in long tape-like formation.

(2) In intestinal dyspepsia with acid fermentation the patient has a jejunal diarrhea, with gelatinous, tenacious, semifluid stools. Mucus is present.

Epithelial or round cells, which are abundant in *catarrhal mucus,* are *absent* from the mucus in dyspepsia of the small intestine. The stools are also green, acid, and give a bile-pigment reaction.

Intestinal parasites may be visible in the feces.

Chemic Examination of the Feces.—*Reaction* is normally *neutral* or *slightly alkaline.* Marked acidity results from occlusion of the bile-duct. Rich vegetable diet causes slight acidity. The simplest method to test the reaction is by litmus-paper (red and blue).

Normal stools react slightly differently with different indicators. With phenolphthalein they react slightly acid, while to litmus they would be neutral. If the phenolphthalein test is employed, take feces 5.0, rub up in a mortar, and add 30.0 cc. distilled water, after the modified Schmidt diet. Place 2 cc. of this in a test-tube, add 2 drops of 1 per cent. alcoholic solution of phenolphthalein. With this quantity titration with decinormal sodium hydrate never exceeds 1.5 cc. to secure end-reaction. Above this the stool should be considered acid, and if less than 1 cc. it may be considered alkaline (Kaplan[1]).

For general use the litmus test is sufficient.

Test for Mucin.—Mucin is normally present in the feces. Mix feces with water and an equal quantity of milk of lime and let the mixture stand for several hours. Then filter, add acetic acid to filtrate, and mucin precipitates if present.

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[1] New York Medical Journal, Dec. 7, 1907.

To Examine Separate Particles of Suspected Mucus.—Dissolve a flake of material in a weak solution of potassium or sodium hydrate and add acetic acid. If the precipitate is undissolved after adding the acid in excess, mucin is present. Heat the precipitate to the boiling-point in a dilute mineral acid; if mucin is present the heated solution will reduce copper oxid. This last test excludes nucleoalbumin, which otherwise gives a similar reaction (Einhorn). Stain a flake of apparent mucus with a weak triacid solution (Ehrlich), mucus produces a green color; albumin, red. This test is of value in determining the presence of mucus in membranous specimens from mucous colic. The tests otherwise are rarely required.

Albumin.—Treat the feces with water slightly acidified with acetic acid. Filter the watery extract and employ boiling test as for albumin in the urine. Normally *no albumin is present*, but it has been found in typhoid, occasionally in acute enteritis, and in chlorosis.

Propeptone and Peptone.—After the test for albumin has proved negative, the watery extract of the feces is treated with phosphotungstic acid, the precipitate is diluted with water and sodium hydrate, and a small amount of a weak solution of sulphate of copper added. A purple red (biuret reaction) shows the presence of both propeptones and peptones. To determine the presence of peptones separately, first precipitate the propeptones by ammonium sulphate in large amount.

Pathologically, peptone is found in typhoid, dysentery, tuberculous ulcer of the intestine, and in perforative peritonitis. Normally it is not present.

Carbohydrates.—The feces are first subjected to distillation. The residue is extracted with alcohol and ether; the extract boiled with water, filtered, and again boiled, with the addition of dilute sulphuric acid. Trommer's or Nylander's test is then employed.

Examination for Starch.—The watery extract of feces is examined with Lugol's solution, the presence of starch producing a blue color.

For Sugar.—A watery extract of feces can be directly tested by Fehling's method.

Normally, neither starch nor sugar are found.

Gas Fermentation.—Schmidt's method will be described later.

Fat.—The feces are treated with considerable ether, and the latter is separated and evaporated in a water-bath. The neutral fat, if present, remains visible.

To show the presence of soaps which do not dissolve in ether, another portion of fecal matter is first treated with acids which split up the soaps, and then extracted with ether; quantitative determination is complicated.

Normally, fat is never present macroscopically in the stools unless after ingestion of very large quantities. It may then be visible in very small portions, the size of a pea. Pathologically, color. Urobilin is normally present and is absent in pathologic conditions, while bilirubin is present in the latter.

Fleischer's Test.—Place a small quantity of feces in a test-tube with a small amount of alcohol to which has been added a few drops of hydrochloric or acetic acid. After a short time urobilin produces a yellow or brown color. If the alcohol is then poured off and a few drops of sodium hydrate with a small quantity of zinc chlorid are added, there appears a green fluorescence in direct rays of light, and in transmitted light, pink or yellowish red, greater or less in proportion.

Acholic and Colorless Stools.—The acholic stool presents a grayish-white, ash-gray, or clay color due to absence of bile-pigment. The dejecta are of penetrating odor, buttery consistency, and on chemic and microscopic examination are found to contain much fat. The latter is present as needle-shaped crystals or in sheaves of crystals or, less generally, in fat-droplets. This type of stool occurs in conditions such as occlusion of the bile-duct, when there is an exclusion of bile from the intestine.

Stools may be entirely *devoid of color* or of a grayish-white color resembling true acholic stools, though there is *no jaundice or occlusion of the bile-ducts.* These stools are less putrid in odor and more acid. They contain enormous amounts of fat, like the true acholic stool, and *urobilin has been demonstrated in them.* Such movements occur in conditions when the absorption of fat is impaired, as in tuberculosis of the intestines and peritoneum. At other times abnormal quantities of fat are not present, and the lack of color is imputed to the presence of a colorless decomposition product of bilirubin, the leuko-urobilin of Mencki.

The conclusion that a stool contains an excessive amount of fat because it is apparently acholic is not justifiable unless a microscopic examination is made. An apparently acholic stool may also be due to excessive fat and urobilin be present.

Fatty Stools (Steatorrhea).—This term is applied to all cases in which isolated masses of fat are present in the feces and can be *recognized with the naked eye.* It appears in whitish or grayish lumps, or it may cling around the feces or be adherent to the vessel.

Ingestion of excessive fat even under normal conditions may produce an evacuation of superfluous fat. If the mucosa of the small intestine and the lymphatic system (mesenteric glands) lose their powers of absorption, fat must appear in the stools, as in tuberculosis of the small intestine, chronic tubercular peritonitis, intestinal catarrh, etc.

In occlusion of bile from the intestines with acholic stools, the fat is revealed microscopically and by chemic analysis, but fatty stools are not then spoken of in the clinical sense.

Steatorrhea is not *per se* diagnostic of pancreatic disease. In the absence of icterus and of demonstrable intestinal disease, fatty stool is probably due to pancreatic disease. Disturbed digestion of fat is diagnostic of pancreatic disease. Müller shows that qualitative changes in the fat (lipolysis) is much slighter, only 39.8 per cent. in pancreatic disease, where it is 84 per cent. in healthy subjects, or even in those with icterus, if the pancreatic juice has free access.

Ferments.—A glycerin extract can be made of the feces or the fecal matter may be mixed with water containing a small proportion of thymol and filtered.

To test for *trypsin*, the filtration extract is made alkaline by the addition of soda bicarbonate and a few flakes of fibrin added. The solutions are kept at a blood temperature for a few hours and then tested with potassium hydrate and a weak solution of copper sulphate. If trypsin is present, there will be a pinkish-red reaction (biuret) in consequence of peptone.

For Diastase.—A few cubic centimeters of the filtrate are mixed with about one-half the amount of a starch solution and kept at a blood temperature for about thirty minutes. The mixture is then subjected to Fehling's or Trommer's test. Normally, these ferments are absent,[1] but in pathologic conditions, especially in diarrhea, they are frequently found. Wynhausen[2] describes a new test for the pancreatic activity. Further investigations are advisable.

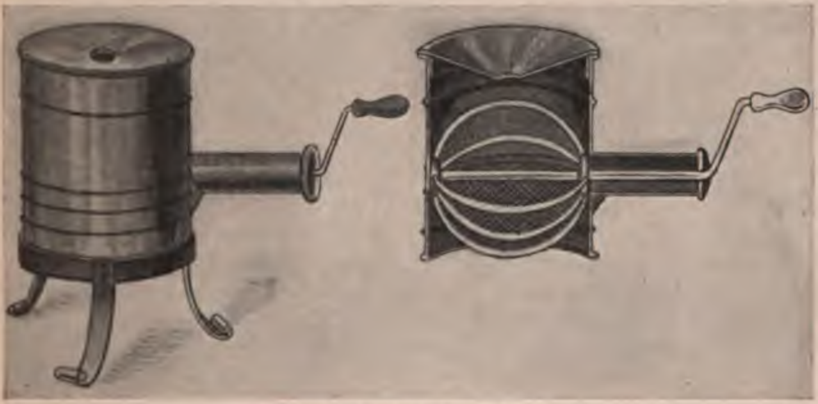

Fig. 176.—Einhorn's stool sieve.

Concretions, Foreign Bodies, Worms.—To examine for such material the feces should be thoroughly mixed with water and poured through a sieve. This can be improvised—a circular wire rim can be fitted to a water-closet seat, and attached to the wire a network bag made of two thicknesses of cheese cloth, practically a dip net. The patient defecates in the net, and water can then be poured through several times until only the more solid parts remain.

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[1] Goldschmidt (Deut. med. Wochenschr., 1909, No. 12, xxv, 522) and Gross (Ibid., 1909, No. 16, xxv, 706), using the latter's method for detecting trypsin, have discovered it in the feces of all normal persons examined. This, if confirmed, will prove a great advance.

[2] Berliner klin. Wochenschr., July 26, 1909; also Med. Record, Sept. 11, 1909.

Boas has constructed a stool sieve and Einhorn's, as in Fig. 176, is readily understood. Water is poured through and there is a stirring apparatus.

Concretions.—Among such are gall-stones, pancreatic calculi, enteroliths, and coproliths. Biliary calculi are readily recognized when of any size. The principal constituents are cholesterin, bile-pigment, and lime.

Tests for small biliary concretions (sand) are as follows: first, powder gr. 30 (2.0) of the mass and treat with ether, ʒv (20 cc.), mix and filter, evaporate, and test for cholesterin. Dissolve part of residue in hot alcohol and allow it to evaporate. Microscopic examination of the precipitate shows rhomboid crystals with ragged edge (cholesterin); second, another part of the residue is placed on a slide, a drop of concentrated sulphuric acid added and covered with a cover-glass, the cholesterin crystals become carmine at their edges, add 1 drop of Lugol's solution and a violet color arises; finally, a portion of the residue is treated with hydrochloric acid, a trace of iron chlorid, and then evaporated. If cholesterin is present, a blue color occurs.

The residue of the original ether mixture is treated with dilute hydrochloric acid mixture, heated, and extracted with chloroform after cooling. The chloroform extract is tested with Gmelin's reaction (fuming nitric acid). Bile-pigment produces the rainbow play of colors.

Pancreatic Calculi.—These usually have a rough surface, are brittle, and may be faceted. They are soluble in chloroform, and on evaporation produce an aromatic odor.[1] Bile-pigment and cholesterin are absent.

Enteroliths.—Calculi formed in the small intestine consist of inorganic salts (lime and magnesia). They are light in color and usually of small size. They form occasionally after the extensive use of lime and magnesia. Rarely they cause obstruction.

Coproliths (fecal calculi) are found in the large bowel, chiefly where there is retardation to the passage of feces, as in the cecum, appendix, sacculi of the colon, sigmoid, and rectum. They are of stony hardness and sausage shape and show concentric rings on section. They may attain considerable size and even cause intestinal obstruction.

Foreign Bodies.—Bodies which have been swallowed may pass through the entire bowel and be passed in the feces, such as bones, coins, marbles, needles, etc. Concretions of shellac have been found in the stools of patients who have drunk furniture polish. Hair-balls may be found.

Microscopic Examination.—The microscopic examination of the feces is often of great assistance to diagnosis. For examination for amebæ, the stool should be kept warm.

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[1] Minch, Berliner klin. Wochenschr., 1898, No. 8.

To diminish the disagreeable odor of a watery stool, place it in a conic glass and cover it with a layer of ether. If it is mushy or firm, it can be spread on a plate and covered with a layer of spirits of turpentine, or a 5 per cent. solution of carbolic acid or thymol, or 4 per cent. formalin.

Diarrheal stools may be examined without further preparation.

With solid fecal matter, a small piece of feces may be placed on a slide and mixed with a drop or two of normal salt solution. If there is odor, a 1 per cent. formalin solution may be added. The findings depend on the diet. With meat diet, no vegetable residue is found, and vice versâ. With a mixed diet, in a normal stool, there will be plant cells, the remnants of various vegetables and fruits, no starch granules, meat-fibers changed beyond recognition, or with slight striation (Fig. 177). More commonly they appear as oval yellow translucent masses with a high degree of refractibility. The presence of numerous meat-fibers striated and with nuclei is pathologic, showing deficient pancreatic digestion (tryptic). They may appear as spirals (Fig. 178).

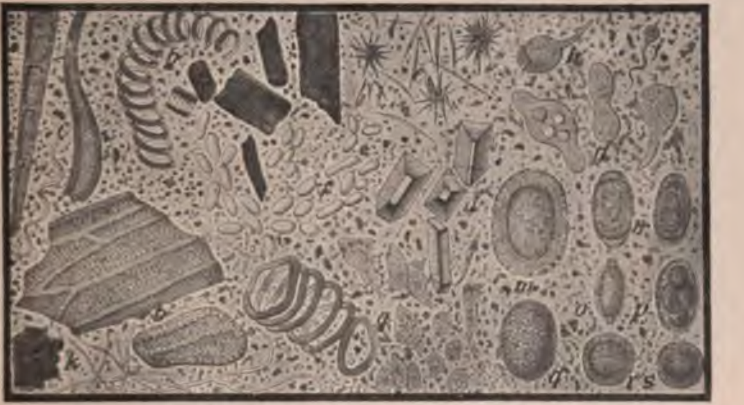

Fig. 177.—General view of the feces: *a*, Epithelial cells and leukocytes; *b*, stone-cells; *c*, cuticular formations; *d*, crystals of ammoniomagnesium phosphate; *e*, fat-crystals; *f*, yeast-fungi; *g*, Amœba coli; *h*, Trichomonas intestinalis; *i*, Cercomonas intestinalis; *m*, ovum of ascaris; *n*, ovum of oxyuris; *o*, ovum of trichocephalus; *p*, ovum of ankylostomum; *q*, ovum of bothriocephalus; *r*, ovum of Tænia saginata; *s*, ovum of Tænia solium (Jakob).

Fat.—Fat appears microscopically as colorless small globules or as needle-shaped crystals (fatty acids) or in sheaves (soaps). The fatty acids disappear when heated or when ether is added; soaps remain unchanged. Sudan dye-stuff, in concentrated alcoholic solution, stains plain fat bright red, while crystals of fatty acid and the soaps remain unchanged. In pathologic conditions these forms of fat are markedly increased, as in affections of the liver, pancreas, and intestines. Normally, they are scanty.

Crystals.—Oxalate of lime, calcium carbonate, neutral phosphate of calcium, and ammonium magnesium phosphate are found in normal as well as pathologic feces and have no diagnostic importance.

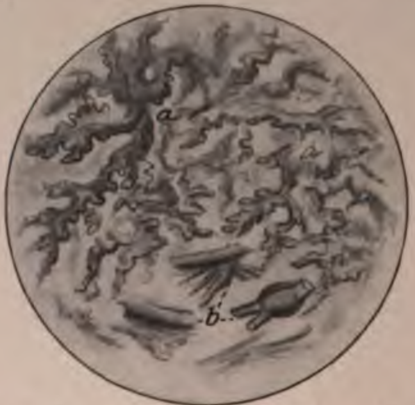

Fig. 178.—*a*, Spirals of undigested meat-fibers in stool; *b*, pieces of bronchi.

Bismuth, if administered, occurs as dark brown or nearly black rhomboid crystals. Hematoidin appears in rhombic crystals of orange or red color, shortly after intestinal hemorrhage. Charcot-Leyden crystals (Fig. 179) are fine colorless, pointed octahedra. These when present excite the suspicion of helminthiasis (intestinal parasites), and their persistence after removal of the tenia shows the

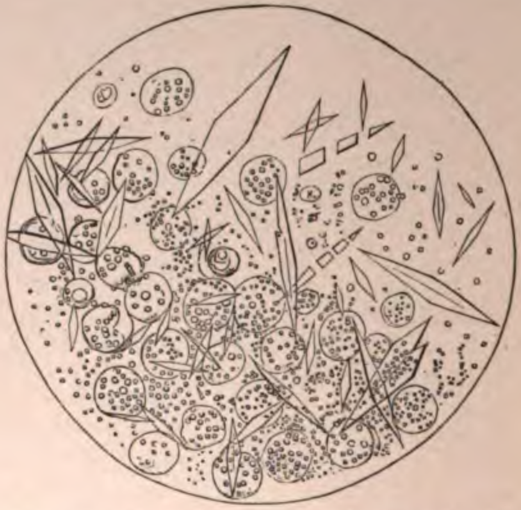

Fig. 179.—Charcot-Leyden crystals (after Riegel).

head has probably not been removed. They occur occasionally in normal stools and in typhoid, dysentery, and phthisis.

Epithelium.—Epithelial cells when present in large numbers always indicate an inflammatory condition of some part of the intestinal tract. Cylindric epithelial cells are found in abundance in inflammation of the intestinal mucosa (Fig. 180). They cause the cloudy appearance of the mucus. If bile-stained specimens of epithelia are met with, the small intestine is involved. Degenerative forms without nuclei are mostly seen, though well preserved cylindric or goblet-cells are often found.

Red blood-cells are rarely observed unless hemorrhage is from the colon or rectum, as in dysentery. Hemorrhage higher up gives a brownish-red color to the feces, and hematoidin rhombic crystals in some cases, and the cells cannot be recognized microscopically.

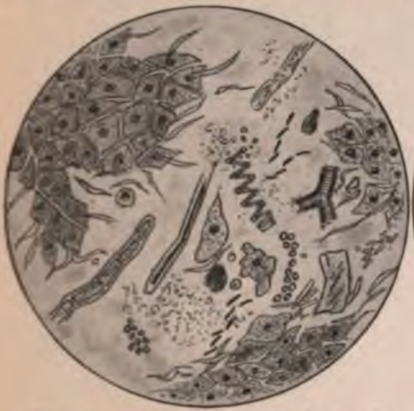

Fig. 180.—Chronic intestinal catarrh: Groups of epithelial cells, detritus, some muscle cells partly digested, bacteria, plant cells, and yeast cells.

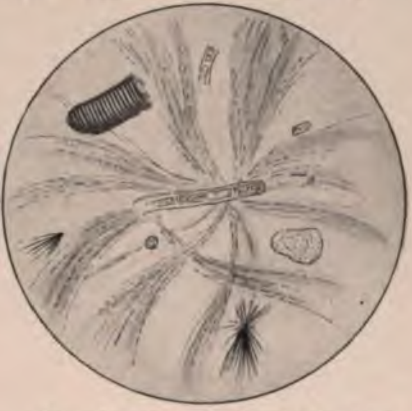

Fig. 181.—Intestinal catarrh: Considerable mucus, some plant cells, muscle cells, and fat crystals.

Pus occurs in the dejecta in ulcerative processes when an abscess communicates with the bowel or in dysentery. It presents the usual characteristics.

Mucus when bile-stained indicates disease of the small intestine; and if colorless, catarrh of the large intestine or lower part of small intestine is present. Mucus also occurs with mucous colic, in which condition no catarrh exists. Mucus is thread-like in appearance, though occasionally amorphous (Fig. 181). Hyaline particles of vegetable residue must not be mistaken for mucus. Iodin stains it blue. Thionin colors it reddish violet and other proteids blue. Mucus has no definite structure.

Tumor.—Fragments of tumor may rarely be found in the feces and its character determined by the microscope.

Microörganisms.—A large portion of the stool is constituted by bacteria, as already mentioned. Among the most important are the

Bacillus coli, Bacillus lactis aërogenes, Bacillus bifidus, Bacillus aërogenes capsulatus (gas-forming), and Bacillus putrificus.

The Bacillus coli is of importance in reference to the indolic type, and the Bacillus aërogenes capsulatus, to the saccharobutyric type of intestinal putrefaction. The Bacillus lactis aërogenes causes fermentation of milk and the production of lactic acid.

The lactic acid producing bacilli are held to be antagonistic to putrefactive changes. For a description the reader is referred to any work on Bacteriology. Typhoid, tubercle, dysenteric, and the cholera bacilli are the chief pathogenic micro-organisms found in the feces which are of interest to us.

TESTING THE INTESTINAL FUNCTIONS

Boas has obtained intestinal juice by passing the stomach-tube into the empty organ and massaging the region of the liver, thus forcing the juice into the stomach. Hemmeter and Kuhn have passed the tube directly. These procedures are uncertain and possess no advantages.

Einhorn[1] has devised a new method of obtaining the intestinal juice by means of a duodenal bucket.[2] The quantity secured (and it is not always obtained) is infinitesimally small; there is the possibility of an admixture of gastric juice and saliva during withdrawal, and the procedure requires three to five hours or even longer. It does not admit of the thorough technic as secured by Schmidt's test diet.

The same holds true of Einhorn's bead test.[3] Testing the motor function with foreign bodies (beads) is not a proper criterion; the food material attached to each bead is too infinitesimally small in amount to test the digestive capacity for an average diet; it may work loose from the beads, in which event it could not be recovered and wrong deductions might result. There is a slight element of risk from fish-bone escaping from the bead and damaging the mucosa.

The author believes *the stomach functions should be tested separately*, and one should not depend on the connective-tissue test for the stomach, as suggested by Schmidt.

I am indebted to E. E. Smith, of New York, for the following description of the tests of the intestinal functions:

Tests of the Intestinal Functions.—The determination of the functional activity of the stomach is made with relative ease, compared to the similar examination of the intestines. Correspondingly, methods for such determination have long been applied to the former organ, while they have recently found application to the latter; and even now the diagnostic determination of intestinal function by exact methods is only in its infancy. The problems presented are essentially these:

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[1] New York Medical Journal, June 20, 1908.

[2] Einhorn has recently suggested passing a soft tube along the duodenal bucket cord (on the principle of Gouley's tunnel sound along the filiform bougie), and then aspirating the intestinal contents with a bulb. Farr (Jour. Amer. Med. Assoc., Dec. 11, 1909) describes Boldireff's oil test meal to secure trypsin in the gastric contents, and the tests of Mett, Volhard, and Gross. These tests are complicated. Trypsin is not always secured.

[3] Med. Record, Feb 10, 1906; Ibid., Oct. 26, 1907; Journal Am. Med. Association, Feb 2, 1907, Therapeutic Gazette, Jan. 15, 1908.

(*a*) Is intestinal secretion, including pancreatic and biliary secretions, normal in quality and quantity? (*b*) Is intestinal absorption normal? (*c*) Is the intestinal motor activity normal? If not, in what respect is each of these processes abnormal?

The investigation of these problems is made by the use of a test diet. While in the case of the stomach the diet is relatively simple and the digestive mixture withdrawn for examination within a few hours, in the case of the intestines only inferences as to the intestinal contents may be reached, and then indirectly from the conditions affecting the bowel contents after they have been ejected, perhaps in one or several days. The feces corresponding to the test diet may be indicated by a material administered for the purpose, usually soot in capsules, No. 00 hard gelatin capsules filled with soot accomplishing this purpose; or carmin may be used, 0.3 gm. (5 gr.), in a capsule. Either of these is administered at the beginning of the first meal of the test diet. The subsequent appearance of a black or red stool indicates that the intestinal contents corresponding to the special diet are being ejected.

The administration of a special substance to mark the stools is in a majority of instances not absolutely necessary, the diet being of a nature that gives rise to a stool sufficiently distinctive for recognition. Until the observer is familiar with the examination, it is advisable to employ one of the substances mentioned.

The test diet to be employed is selected to meet the requirement that it shall present a sufficient quantity of *all classes of food stuffs to test the digestive capacity*, that the digestive processes shall not be unduly anticipated in the preparation of the food, and that very little food residue shall be present in the ejected bowel contents. Naturally, the stomach plays its usual part, so that the test is not limited to the intestinal tract proper, but applies to alimentation as a whole.

The test diet made use of is a modification of the original Schmidt-Strasburger diets. Instead of three diets, as were originally employed in connection with the fermentation test of these observers, one diet is now advocated which presents the conditions essential for the meat test. This diet, as recently described by Schmidt, is as follows:

In the Morning.—0.5 liter milk or, if milk does not agree, 0.5 liter cocoa (prepared from 20 gm. cocoa powder, 10 gm. sugar, 400 gm. water, and 100 gm. milk), to this add 50 gm. Zwieback.

In the Forenoon.—0.5 liter oatmeal gruel—made from 40 gm. oatmeal, 10 gm. butter, 100 gm. milk, 300 gm. water, 1 egg—(strained).

At Noon.—125 gm. chopped beef (raw weight), broiled rare with 20 gm. of butter, so that the interior will remain raw; to this add 250 gm. potato broth (made of 190 gm. mashed potatoes, 100 gm. milk, and 10 gm. butter).

In the Afternoon.—As in the morning.

In the Evening.—As in the forenoon.

This diet consists of:

1.5 liters milk,
100 gm. Zwieback,
2 eggs,
50 gm. butter,
125 gm. beef,
190 gm. potatoes and gruel of
80 gm. oatmeal.

It contains about:

102 gm. albumin,
111 gm. fat,
191 gm. carbohydrates, or a total of 2234 calories (raw calories).

The test is generally given for three days, sometimes longer; at any rate until a stool is obtained, which comes with certainty from this diet.

Steele advocates an arrangement of the diet to conform to American dietary habits, which still maintains the essential features of the above. It consists of:

2¾ pints milk,
3 ounces well-dried toast,
2 eggs,
1½ ounces butter,
¼ pound tender rare steak,
6 ounces mashed boiled white potato, and gruel made from
2½ ounces ordinary oatmeal,
½ ounce sugar.

This may be given somewhat as follows:

Breakfast.—2 eggs, one-third of the amount of toast and butter, 2 glasses of milk, oatmeal, and sugar.

Dinner.—The steak and potato, one-third of the amount of toast and butter, 1½ glasses of milk.

Supper.—2 glasses of milk, remainder of toast and butter.

For the collection of the stools, where they are not to be transported for any considerable distance, an ordinary tin basin of 24-oz. capacity serves well for a receptacle, as suggested by Prof. L. B. Mendel. This may be supplied with a cover, consisting of a cake tin of appropriate size; or, where this is not readily provided, a small pie tin may be used. This outfit is inexpensive (8 cents), adequate, and where a considerable number are to be kept, may be advantageously stacked. Moreover, where the stools are to be weighed and dried for exact quantitative analysis, this may be directly done in the weighed basins, thus avoiding the transfer of the specimens.

If the feces are to be transported for any considerable distance, a pint glass jar (with patent airtight top) serves admirably for the collection. The specimen may be hermetically sealed. If transported in ordinary wide-mouthed bottles, the stoppers should be tied on, as gas formation is quite likely to produce sufficient pressure to force out any stopper not securely fastened.

The characteristics of the test diet stool, aside from the coloration given by the special marking substance administered, are the light brown color and uniform consistency. It usually appears at the second or third defecation after the beginning of the test diet.

The *period of time required* for the passage of food through the entire alimentary tract is of importance and is readily observed. Normally it takes about twenty-four hours. It is not necessarily, though it is commonly, related to the frequency of defecation. In some cases the stool appears with regularity and is fairly copious, yet the patient suffers from fecal accumulation—a latent constipation. The period of passage may suggest the seat of the intestinal disturbance in diarrhea, since it is only decidedly increased when the cause is high up. Strauss has shown that chronic colitis may be accompanied by several watery movements a day with a normal period of passage. The examination of the collected stool should be made while the feces are perfectly fresh.

Macroscopic Examination.—This is the most important part of the procedure. Experience *with this part of the investigation* may enable the observer *to at once recognize some defect in alimentation.*

Note the consistence, color, and odor. Inspect the surface of the formed stool for morbid products, notably for pus, blood, and mucus, which are to be removed for microscopic and bacteriologic examination. Bits of tissue from some diseased area may also be sought for, but their occurrence is so unusual that it is exceptional when they are found.

The mass of feces is then well mixed, for which procedure a wooden or tin-plate spoon is useful; and a piece the size of a walnut transferred to a mortar, in which it is thoroughly but not forcibly ground with distilled water added gradually until the whole is of a uniform fluid consistency and no small masses of fecal matter remain. The fluid feces is then examined in thin layers, conveniently in a Petri or similar larger dish, against a black background with the naked eye, or low-power magnifying glass, for all elements that may be differentiated. In normal digestion only a few brown points smaller than pin-heads will appear, these consisting of chaffy remains of oatmeal gruel and remains of cocoa, if this latter has been taken. Under pathologic conditions, there may be present:

1. Mucus, which appears usually as larger or smaller, soft, glossy, translucent flakes, often bile stained; infrequently, when from the large intestine, white or brown, with a gummy or almost leathery hardness.
2. Pus, blood, parasites, stones, and other foreign bodies.
3. Connective tissue and tendons, distinguished by their whitish-yellow color, thread-like appearance, and solid consistence.

4. Muscular tissue, chiefly in very small, brown colored rods, like splinters of wood.

5. Potato, appearing like boiled tapioca grains, readily confused with flakes of mucus. The distinction is made with the microscope.

6. Large crystals of ammoniomagnesium phosphate, which grate when the specimen is ground.

Microscopic Examination.—This serves chiefly to complete the gross inspection. In addition to the preparations of material selected during the macroscopic examination, three slide preparations are made. The *first* consists merely of a drop of the liquefied stool under the cover-glass. The *second*, a drop of the liquefied stool mixed with a drop of acetic acid, heated to the beginning of boiling and covered with a cover-glass. The *third* consists of a drop of the liquefied stool mixed with potassium iodid solution of iodin and covered with a cover-glass.

Inspection of the first slide preparation by the aid of the microscope, using a high, dry lens, reveals finely divided material consisting of bacteria and mostly unrecognizable detritus, in which are imbedded:

(*a*) Isolated fragments of muscle-fibers, usually bile stained, partially digested, but occasionally with the transverse striations recognizable.

(*b*) Larger or smaller yellow crystals of the alkali earth salts of the fatty acids.

(*c*) Colorless soaps.

(*d*) Isolated potato cells, without distinguishable contents.

(*e*) Particles of oatmeal and cocoa shells, where the latter is taken instead of milk.

The second slide gives a general idea of the fat present in the stool. While hot, the fatty acids, liberated in the acetic acid, appear as drops; on cooling, these congeal to small needle-like crystals.

In the third preparation potato remains have a violet color, while isolated fungus spores (Clostridium butyricum) may appear blue.

The pathologic findings which the slide may present, in addition to those enumerated under the macroscopic examination, are:

Slide I.—Muscle fragments in greater number and better state of preservation, particularly with retained nuclei; drops of neutral fat; needles of fatty acids and soaps; many groups of potato cells.

Slide II.—Massive fatty acid drops and crystals.

Slide III.—Blue starch grains, free or in the potato cells; oatmeal cells; any considerable number of blue-staining fungus spores or thread-like bacteria.

Bacteriologic Examination.—The recognition of the tubercle, typhoid, Shiga, or cholera bacillus calls for technic which will be found described in special treatises. The selection of material to be examined for tubercle bacilli is best made from the surface of formed stools, since in soft movements morbid products from the diseased area will be so mixed with the feces as to easily escape detection.

For amebæ a saline[1] should be given and the warm liquid stool examined. Mention has already been made of the detection of bacteria and fungi which are colored blue by iodin (granulose reaction).

Procedures that yield information of considerable value in the less specific forms of intestinal infection have recently been advocated by Herter. Of first importance is the preparation of smears of the mixed stools on microscopic slides, stained by the Gram method. The relative number of Gram-positive bacteria, as also their character, is of diagnostic value, since they are relatively few in health and in meat-free diet, while they are increased in some diseased conditions and when notable quantities of meat are eaten. Not only is it of value to note the actual increase, but the potential. The latter is determined by the observation of the relative number of Gram-positive bacteria in the residues in the sugar-bouillon tubes (see below), a predominance of Gram-positive bacteria indicating a pathologic tendency of the fecal flora.

The sugar-bouillon tubes just mentioned are ordinary fermentation tubes, containing 1 per cent. lactose-bouillon, 1 per cent. glucose-bouillon, and 1 per cent. saccharose-bouillon respectively.

Normally, little fermentation occurs when the tubes have been incubated at 37° C. for twenty to twenty-two hours. Active gas production, so that it accumulates to the extent of more than one-third of the tube capacity, is most frequently due to the predominance of a bacillus identical with or allied to the Gram-positive Bacillus aërogenes capsulatus, the growth of which replaces the Bacillus coli communis, the normal intestinal inhabitant.

Chemic Examination.—The *chemic reaction* is best determined by smearing one side only of moistened red and blue litmus-paper with the diluted (see Macroscopic Examination) fecal matter. After some time the reaction is noted on the opposite side. It is usually amphoteric, feebly acid, or alkaline.

The *sublimate test* for unchanged bile-pigment is performed by adding some of the diluted fecal matter to a considerable excess of strong solution of bichlorid of mercury, allowing the mixture to stand over night. The normal feces are colored red; more intensely, the fresher and less decomposed the excrement. Herter points out that this red coloration may be pathologically increased in excessive saccharobutyric putrefaction. *In the presence of bilirubin, a green coloration is produced.* This, even to the extent of microscopically small particles, is pathologic. A negative sublimate test suggests suppression of bile; an incomplete test with fresh stools, abnormal processes of decomposition.

The *fermentation test* of Schmidt and Strasburger for fermentable carbohydrate or putrescible proteid is performed in the Strasburger fermentation tube (Fig. 182).

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[1] Magnesium sulphate is an excellent saline cathartic to employ.

A 5-gram portion of the well-mixed, undiluted, fresh excrement, or proportionally more of the thinner material, is well mixed with sterile water; the chemic reaction noted; and then the mixture is introduced into the lower vessel of the apparatus.

The bottle is entirely filled with water and stoppered, with exclusion of air. The adjacent upper tube is filled or nearly filled with water, while the distal tube remains empty. The apparatus is incubated at 37° C. for twenty-four hours.

The extent of gas formation is indicated by the amount of water displaced by the gas from *b′* and which accumulates in the distal tube *c′*. Normally, there is practically no gas formation, and the chemic reaction of the fecal mixture remains about unchanged. Gas production to such an extent as to introduce an amount of water into the distal tube equal to one-third its capacity is pathologic. If coincidently with the gas production the chemic reaction has developed a decidedly increased acidity, the gas production is due to carbohydrate fermentation; if alkalinity, albuminous putrefaction has occurred. When the proximal tube is opened it gives off a butyric acid odor in the former case and a putrefactive odor in the latter. The color of fermenting feces is generally brighter; of putrefying feces, darker. The test is more especially applicable to the test diet stools.

If the condition approximates the normal, a further test should be carried out for accuracy. In such event, the patient is placed on a diet which differs only from the first in the absence of meat and potato. If there is still a positive result, the diagnosis of "fermentative dyspepsia" is justifiable.

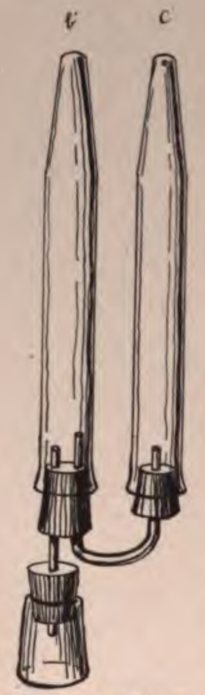

Fig. 182. — Strasburger's tube.

Putrefactive Products.—Tests for indol and skatol may be applied to the distillate, using 10 gm. of the feces mixed with 120 cc. of water, the whole made alkaline, a bit of paraffin added to prevent frothing, and the first 50 cc. collected.

A suitable apparatus for conducting the process consists of a 500-cc. capacity long-neck Kjeldahl digestion flask connected with a Liebig condenser. Distillation with steam is sometimes advantageous; 10 cc. of the distillate is rendered slightly alkaline with sodium or potassium hydroxid and an excess of a fresh solution of beta-naphthaquinone-sodium-monosulphanate added. The substance, in the course of a few minutes, reacts almost completely with the indol present, but not with the skatol; with the resulting formation of a bluish precipitate with much indol, and a mere coloration of the solution with little. If more than a trace is present, the reaction is conducted with the remaining 40 cc. of the distillate, the indol-naphthaquinone compound removed from the whole by filtration, and from the portion remaining in solution by distillation, after acidifying.

The distillate containing the skatol, if necessary, freed from indol as described, is tested by the use of a well-marked excess of dimethyl-amido-benzaldehyd (Ehrlich's aldehyd), being boiled with a 5 per cent. solution in 10 per cent. sulphuric acid. Dilute hydrochloric acid is added to the point of the production of the maximum color intensity and the mixture rapidly cooled. The presence of skatol is indicated by the blue coloration. The color may with advantage be extracted with chloroform.

If the process described is to be conducted quantitatively, 25 gm. of feces should be employed, and distillation continued till the distillate is free from substances reacting with the above reagents, the color shaken out with known volumes of chloroform, and the depth of color compared by the aid of the Duboscq colorimeter, with a similar chloroform extract obtained by starting with solutions of known strength of indol and skatol respectively.

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4. Steele, J. Dutton, Medical News, 1905, p. 1158, vol. lxxvii.

Results.—In interpretation of results, we have to consider both the occurrence of pathologic admixtures and an increase of the constituents from the test diet.

A thin coating of mucus is normally collected by the hardened fecal matter. Otherwise the appearance of this product indicates catarrh of the mucosa, the mucus containing many cellular elements. An exception is the overproduction of mucus in colica mucosa, with cellular elements less abundant and with the striking consistence.

Pus, blood, masses of epithelia from the mucosa, and similar elements carry the pathologic significance of these products, and require investigation and interpretation, as when found elsewhere.

Considering an increase of the constitutents of the test diet, a distinct connective-tissue increase indicates deficient gastric[1] digestion. An excessive quantity of meat-fibers points to deficient proteid digestion in the small intestine. The albumin fermentation (putrefactive) test further indicates increased proteid in stools, frequently derived from pathologic secretions, probably associated with an abnormal putrefactive flora. The bacteriologic examination, especially as to fermenting and Gram-positive bacteria, is of value along these lines. Also tests for putrefactive products, if increased activity in this direction in the stool itself is to be investigated.

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[1] The author advocates a separate test of the gastric functions.

Starch granules, revealed microscopically, show deficient starch digestion in the intestines, due either to rapid passage of the contents or disturbed secretion. Defective carbohydrate digestion is also indicated by an abnormal carbohydrate fermentation test.

Only a considerable increase of fat is pathologic. Such a stool is indicated by the quantity of the stool itself, the light (whitish) color, and a marked acid reaction, as well as by the microscopic findings. A deficient flow of bile or of pancreatic secretion is the usual cause; in the former case the stool not containing pigment (sublimate test); in the latter showing an associated increase of meat-fibers, frequently with retained nuclei. Functional disturbances of fat digestion are said to occur, but other possibilities must be excluded in reaching this diagnosis.

MECHANICAL PROCEDURES

The Enema; Intestinal Irrigation; Proctoclysis.—For injection into or irrigation of the intestines there are *four methods*, all of which have their special applications. They are:

1. The enema.
2. Irrigation with a single tube.
3. Irrigation with a double-current tube or with two tubes.
4. Proctoclysis, the drop method of injection.

Uses of irrigations or enemata are as follows: The local treatment of diseased conditions, as of catarrhal colitis.

The relief of congestion or acute inflammation, as of the rectum or prostate.

The relief of pain and irritability, as in spasm of the sphincter.

The absorption of inflammatory products, as of postuterine adhesions.

To replace the loss of fluid in the body, as in cholera.

To dilute the poison of disease, as in uremia.

To increase the flow of blood to a part, as in insufficient menstruation.

To check hemorrhage (extreme cold or heat). Locally, as in bleeding ulcers of the rectum; in an adjacent organ, as in uterine hemorrhage.

Reflex effects through the sympathetic ganglia, on the circulatory apparatus, on the secretions, as a tonic stimulant, and the revulsive effects. On the circulatory apparatus, as in shock. On the secretions, as in the production of sweating, bowel action, and urinary secretion in uremia. As a tonic stimulant, as by use of the alternate hot and cold douche in diminution of erectile power. The revulsive effect, as the production of bowel action in apoplexy. The reflex effect on a distant organ, as from enemata in jaundice.

The effect on the heat centers—the temperature can be raised in shock by hot irrigation, or lowered in fever by cold irrigation or by enemata.

Simple cleanliness, removing undigested food products, and preventing auto-intoxication.

Antispasmodic, as by relieving spasm in colic.

Mechanical, as in intussusception.

Water as a vehicle—the nutritive enema.

Physiologic Experiments.—In a series of experiments at Columbia University some years ago the author demonstrated on animals (Fig. 183), and later clinically, that enteroclysis at 110° to 120° F. best stimulated the heart in shock; that renal secretion undergoes a double cycle of increase from enteroclysis at high temperatures (110° to 120° F.), both from intestinal absorption and from the increased blood flow through the kidney; while with lower temperatures the increase is merely from intestinal absorption.

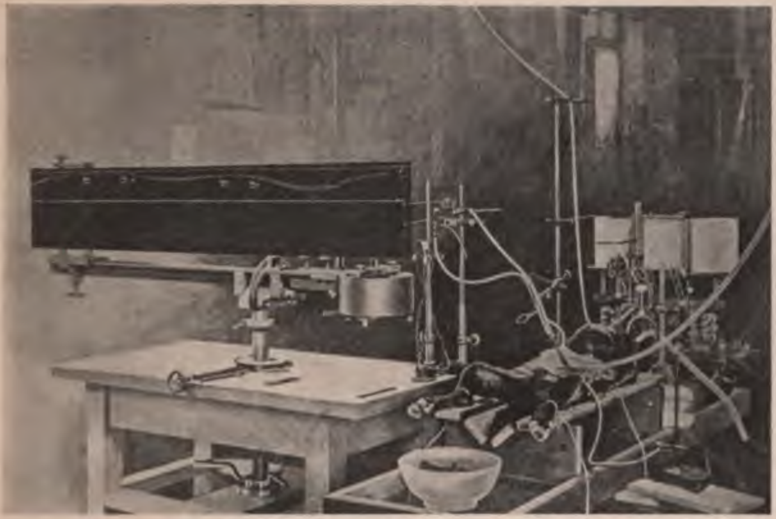

Fig. 183.—Method of performing physiologic experiments.

Normal saline solution has a specific effect in increasing renal secretion. Cold irrigations first stimulate, then depress. Body and blood temperature are increased by hot irrigations and diminished by cold.

These experiments[1] were completely reported.

1. *Enema.*—The enema *may be high with a colon-tube* (Fig. 184), which should be thoroughly lubricated and the water should flow while inserting. It should be administered with the patient on the left side,[2] or for high injection in the knee-elbow posture, when a

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[1] Enteroclysis, Hypodermoclysis, and Infusion, Kemp; Hydrotherapy, S. Baruch; Enteroclysis, Reference Hand-book of the Medical Sciences, 1900.

[2] The dorsal position, with the hips elevated, can be employed.

high enema can thus be given with a short tip. I prefer never to give more than 1 to 1½ quarts (liters).

Fig. 184.—Colon-tube.

Milder medicated solutions can be employed in this way. The low enema is of more value for low impaction or to relieve local irritation in the rectum or adjacent organs.

2. *Irrigation with a Single Tube.*—There are four modifications of this method:

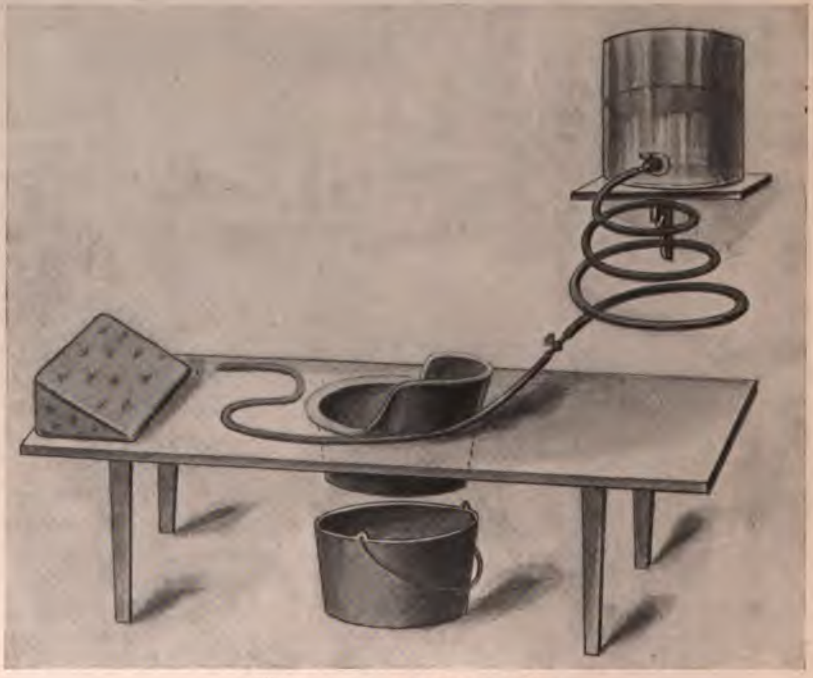

Fig. 185.—Lee's cholera table.

(*a*) A colon-tube is inserted into the bowel, a funnel attached, and by raising and lowering the funnel, the bowel is washed out.

(*b*) A fountain syringe can be attached to the colon-tube, and when sufficient fluid has flowed in the fountain syringe the connection is detached and the fluid flows out through the colon-tube.

(*c*) The patient can void the fluid around the colon-tube or catheter during irrigation. This is the method usually employed with infants. Elmer Lee carried out this technic at the cholera stations (Fig. 185). An irrigating jar may be substituted for the rubber bag.

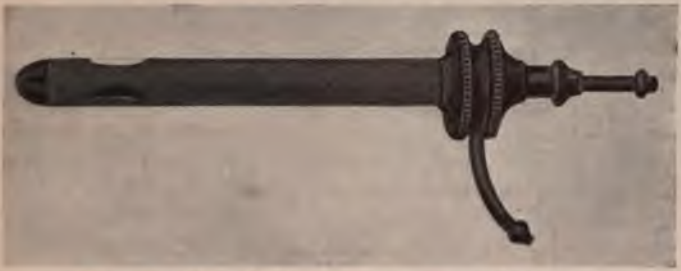

Fig. 186.—Kemp's flexible recurrent rectal irrigator.

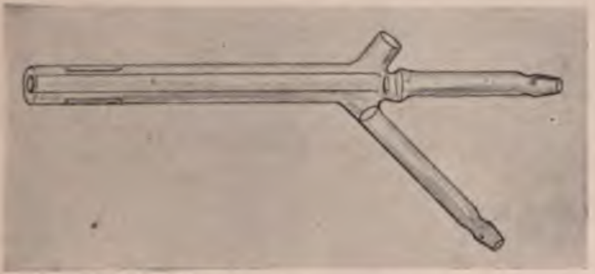

Fig. 187.—Kemp's glass rectal irrigator (recurrent). Cork opening above for cleansing.

(*d*) A glass Y is attached to the colon-tube, as in lavage of the stomach; one branch is connected with the fountain syringe, the other is to a carry-off tube. By alternately pinching the soft-rubber outflow and inflow tubes the bowel can be irrigated.

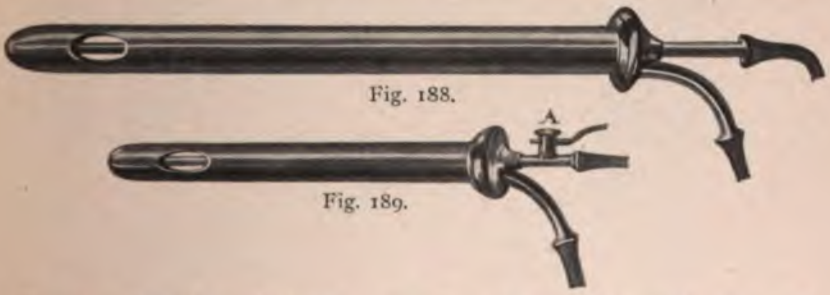

Fig. 188.

Fig. 189.

Fig. 188.—Kemp's rectal irrigator. All metal tube.

Fig. 189.—Electric attachment for electro-enteroclysis. Hard-rubber tube with metal center.

3. *Double-current Irrigation with Two Tubes or a Recurrent Tube.—Advantages.*—The quantity of the fluid is under the control of the operator, since it can be regulated by manipulation of the outflow and inflow tubes.

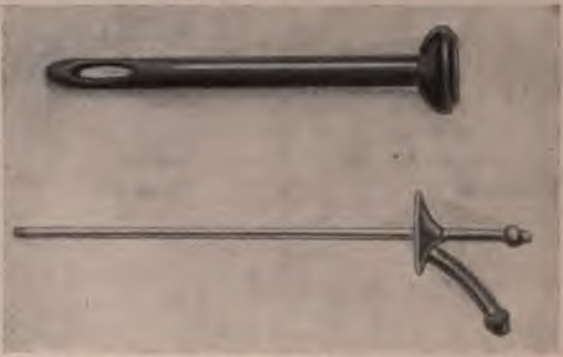

Fig. 190.—Kemp's tube (ready for cleansing). Hard rubber with metal center.

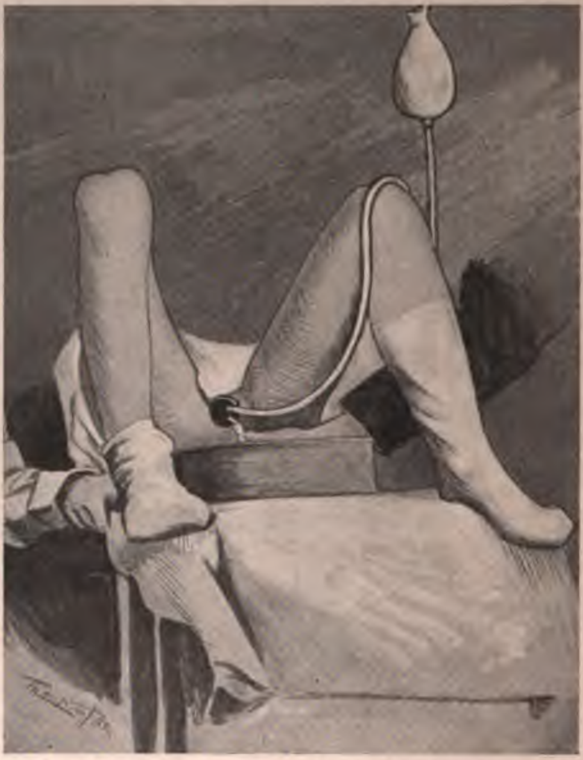

Fig. 191.—Enteroclysis (double current). Patient in dorsal position on bed-pan.

The labor is placed upon the operator and not upon the patient, and there is no straining to overcome the resistance of the sphincter. The straining of self-evacuation is avoided, and mere mechanical cleansing of the bowel is employed. The temperature of the fluid entering the bowel can be kept constant.

Tympanites is relieved best by this method, the return flow carries off the gas by suction. With the enema the gas frequently collects in the intestines behind the injection and it is often impossible to exert sufficient force to expel it with the enema.

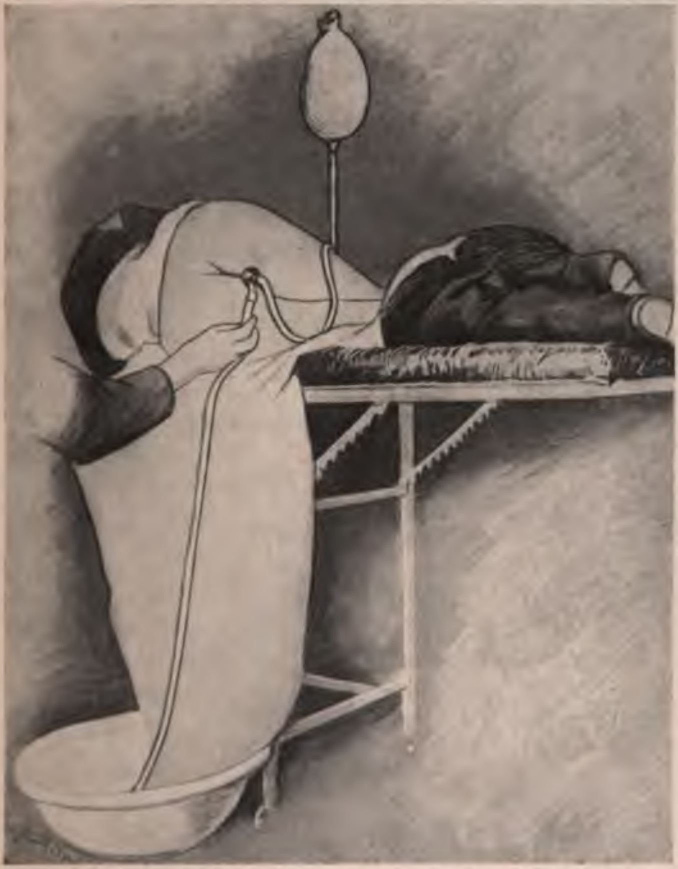

Fig. 192.—Enteroclysis (double current). Patient in Sims' position.

Two catheters or two small rectal tubes passed through a perineal pad can be improvised for this purpose. The illustrations of the author's tubes are shown (Figs. 186–190). They are readily understood. J. P. Tuttle's, Hemmeter's, and various recurrent rectal tubes are described by the author in his manual "Enteroclysis." The hard-rubber tube with metal center is the best. All metal tubes are good for hospital work.

The flexible tube is excellent for sensitive cases or for young children.

To the middle tube of the irrigator is attached the tube of the fountain syringe; to the curved tube the outflow soft-rubber tubing is fastened. This last must be pinched, as it is the larger, in order that fluid may pass up the bowel. By alternately pinching the inflow and outflow the quantity of fluid may be regulated.

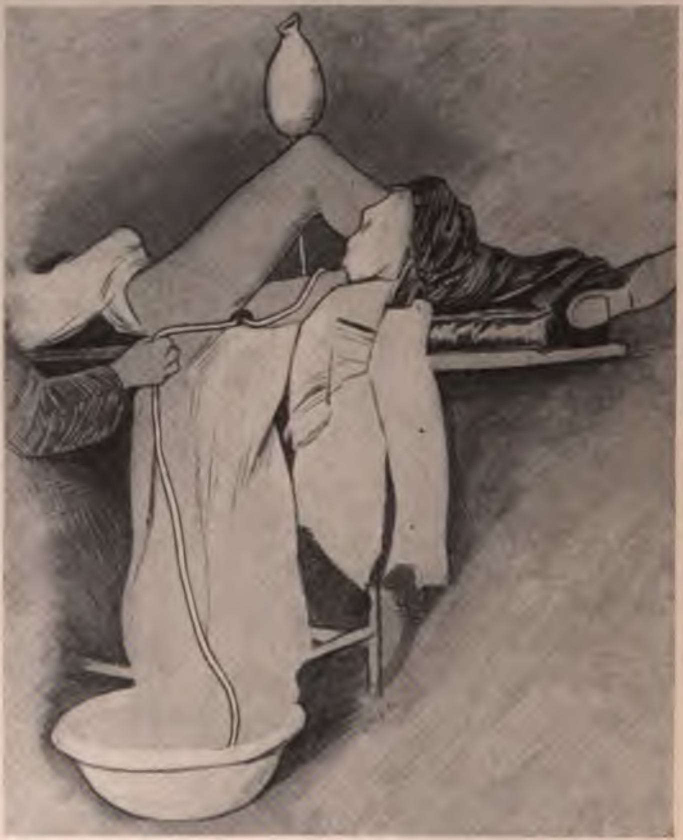

Fig 193.—Enteroclysis (double current) without the bed-pan.

Position of the Patient.—Elevation of the hips is the important feature, not the length of the tube. The different positions of the patients are illustrated and are readily understood (Figs. 191–195).

Method by Rotation.—Patient is placed on the left side with the hips elevated, and the descending colon is irrigated.

Rotate the patient gradually to the dorsal position, and then to the right side, with the hips elevated; the return tube being pinched. About 1½ pints to 1 quart (750 to 1000 cc.) of fluid are allowed to run into the bowel.

The shoulders are then elevated to above the level of the hips, the patient being still on the right side. This is to make the fluid gravitate into the caput coli.

The shoulders are then depressed to below the hip level, the patient on the right side; he is then gradually rotated to the dorsal position and then to the left side, and as a final step the shoulders are elevated, etc. In other words, the process is reversed. The return tube is then released and the fluid is allowed to escape.

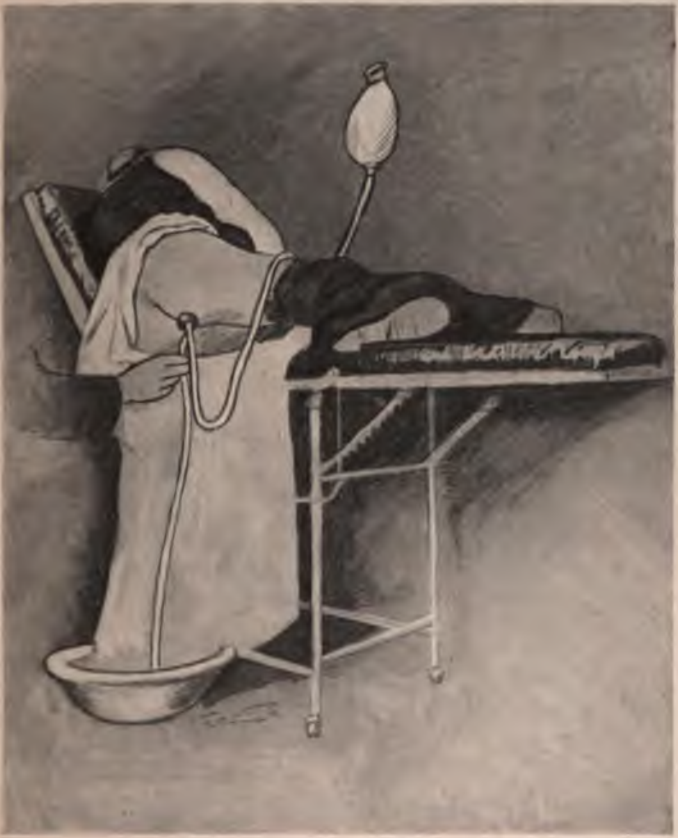

Fig. 194.—Enteroclysis (double current). Patient in semi-oblique position, as in pulmonary edema, etc., when dorsal position is impossible.

I employ *most frequently the dorsal position without the bed-pan.*

Temperature of the solution depends upon the conditions of its employment, an average of 101° to 105° F. in intestinal catarrh; at 110° F. in typhoid for an additional eliminative effect, and for shock and uremia at 115° to 120° F.

Solutions Employed.—Flaxseed tea (2 drams—8.0—of flaxseed to 1 quart—liter—of water, boiled twenty to thirty minutes and strained). Temperature, 101° to 103° F. This should be rather thin and oily in order to flow easily from a fountain syringe. Dilute it with boiled water if too thick. Normal saline solution with oil of peppermint, ♏v to xv (0.296–0.88 cc.), or oil of cinnamon, ♏v to xv (0.296–0.88), to 1 pint (500 cc.). Listerin, borolyptol, glycothymolin, borax, bicarbonate of soda, tannic acid, and alum have been employed at a strength of ʒj (4.0) to 1 quart (liter).

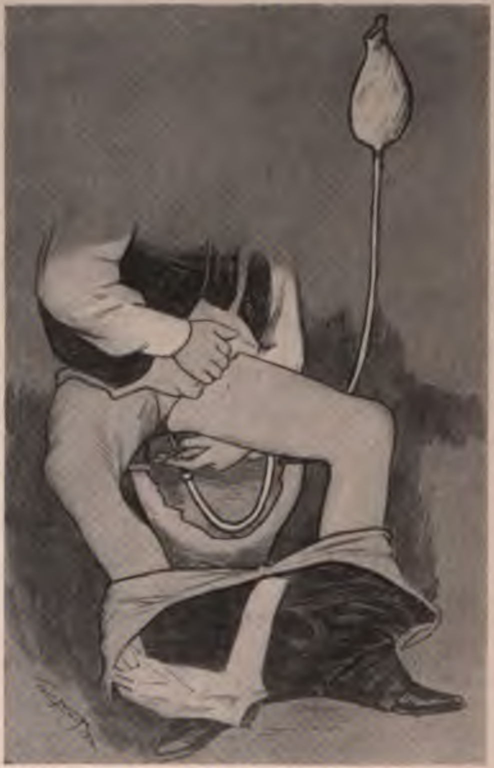

Fig. 195.—Enteroclysis (double current). Self-irrigation of the prostate per rectum.

Delafield has used bichlorid of mercury (1 : 10,000), 2 quarts (liters), for septic membranous colitis complicating typhoid fever, with a recurrent tube, with success. Gum-arabic solution is often of service. Special solutions are described appropriately.

Normal saline solution, ʒj (4.0) of salt to 1 pint (500 cc.) of water, has a wide field of usefulness.

Enemata and enteroclysis are of value in dysentery, intestinal hemorrhage,[1] intestinal catarrh, typhoid, intestinal colic, tympanites, intestinal toxemias, thirst, constipation, impaction, intestinal paresis, and jaundice.

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[1] This refers to hemorrhage in the large intestine, where an extremely hot or cold astringent enema is of service.

They are extremely useful in shock, uremia, sepsis, renal colic, and in inflammatory conditions of the genito-urinary organs of both sexes.

Proctoclysis.—In conclusion, I wish to refer to proctoclysis, the injection of normal saline solution into the rectum by the drop method, as first suggested by Dr. John B. Murphy of Chicago. This procedure is of special value in sepsis, and is of use as an adjunct to other treatment in postoperative shock, intestinal paresis, and uremia. In my experience the employment of continuous (recurrent) irrigation, with a temperature of the saline solution at 120° F., is more efficacious in the latter conditions, followed by proctoclysis as an adjunct.

One of the difficulties which the physician must endeavor to overcome in the administration of saline solution by the rectum or by infusion is the maintenance of a constant temperature of the solution. Elbrecht's apparatus necessitates a special heating chamber in addition to the containing reservoir, with the employment of an electric heater, an alcohol lamp, or a Bunsen burner. The method, though scientific, seems complicated and is quite expensive.

I have recently employed the vacuum bottle[1] with a specially devised attachment which has proved efficacious in preserving the saline solution at a constant temperature. The device is readily understood from the illustration (Fig. 196).

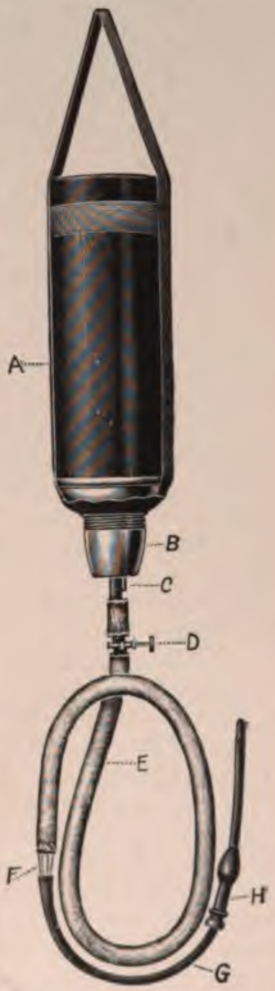

Fig. 196.—Heat-retaining proctoclysis bottle.

Through the screw cap (*B*), which closes the bottle, passes a small hard-rubber conducting tube, to which is attached the outflow tube (*E*). Parallel with this is the filiform tube (*C*), which allows the entrance of a fine column of air, so as to render the flow possible. This last tube passes through the solution to within about ⅛ inch from the bottom of the bottle. As the instrument is employed inverted, it would correspond to the same distance from the top of the bottle. This filiform tube is of hard rubber externally where exposed to the air as a non-conductor of heat. The part lying within the bottle is purposely made of metal, so that it is rapidly heated by the surrounding solution and the entering air is thus, in turn, heated markedly.

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[1] New York Medical Journal and Philadelphia Medical Journal Incorporated, Aug. 14, 1909.

A series of experiments have demonstrated that there is only a loss of 1° to 2° F. in the temperature of the solution in the bottle during the administration of proctoclysis (the drop method) lasting half an hour, a negligible amount. The screw compression valve (*D*) is applied close to the bottle attachment, so as to avoid as much as possible the solution cooling in the soft outflow tube. This outflow tube (*E*) is joined to the catheter (*G*) by a short piece of glass tubing (*F*), for the purpose of observing whether the flow is constant. The catheter for rectal injection passes through a self-retaining rectal tip (*H*), and the former can be inserted to any length desired. The conducting tube (*E*) is especially thick, as in Elbrecht's apparatus.

An asbestos tube surrounds the conducting tube from its junction at the bottle to the catheter. This lessens dissipation of heat and obviates the use of hot towels. The asbestos wrapping can be occasionally slipped off the glass connecting joint, so as to observe the flow. The vacuum bottle is filled in the usual manner and the special cap with attachment screwed on. The bottle is then inverted and suspended in a cord sling, as in the illustration. A small amount of fluid will escape from the bottle by the filiform air tube until the solution reaches the level of the tube, which now lies near the top of the bottle. The bottle is then suspended about 6 inches above the rectum or higher if desired, and the flow tested for the proper speed before inserting the rectal tip and catheter.

If flatus occur, lower the reservoir for a brief period to below the level of the abdomen, so the gas may escape into the bottle. At times, however, it may be necessary to remove the tube for a short period. The insertion of a T-tube between the reservoir tube and rectal catheter is of advantage. A short piece of rubber tubing is attached to the branch and immediately clamped. On occurrence of flatus, the lateral clamp is removed and the gas allowed to escape. It is then reclamped. This obviates removal of the tube. As already stated, there is practically no loss of heat in the container, all of it occurring during the passage of the drops through the outflow, tube; the slower the speed, the greater the loss.

At the start the speed is always more rapid and, though gauged to say 15 drops per minute, may in the course of two minutes drop to 5. A test of two to three minutes should, therefore, be made before inserting the catheter, so as to insure a constant flow at the desired rate. The following table will be found of service; with

Temperature of water in bottle.	Length of tube (inches).	Number of drops per minute.	Temperature in rectum.
160° F.	30	20 or less	100° F.
150° F.	30	40 to 50	100° F.
138° to 140° F.	30	150 to 200	105° to 110° F.

If the injection is given at a greater speed than 200 drops per minute, the solution in the bottle should not be over 120° F., as there is practically no loss of temperature. This method by enema or recurrent enteroclysis would be of great value in shock. It could then be followed by proctoclysis as an adjunct.

Hypodermoclysis.—There is a loss of 10° to 20° F. during the injection, depending upon the size of the hypodermic needle.

Infusion.—Dawbarn advocates a temperature of 115° to 120° F., preferably the latter; time, ten minutes to the liter (quart).

With the smaller vacuum bottle, containing about 1 quart (liter), a glass V tube can be inserted between the conducting tube and the rubber tube for attachment to the infusion canula. By this means it is possible to tell when the bottle is empty and thus prevent the entrance of air. A clamp can be applied close to the V tube on the canula side and the bottle refilled, the V tube being refilled before the conducting tube is reattached, and the latter being done while the solution is flowing.

A larger bottle can be secured for infusion, but the smaller one can be employed with these precautions.

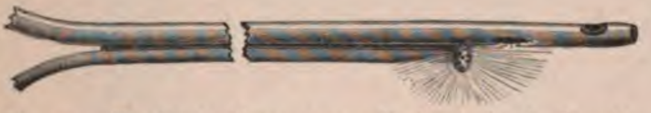

Fig. 197.—Türck's double-current needle douche for the sigmoid.

The temperature of the saline solution does not practically change during the infusion and should be at 115° to 120° F. in the reservoir.

Needle Douche; Nebulizer; Colonic Massage Bags; Gyromele.—These instruments[1] have been devised by F. B. Türck. The colon needle douche (Fig. 197) I believe of some value for local treatment by the alternate hot and cold spray in the atonic type of constipation.

The nebulizer (Fig. 198) is recommended by its inventor for spraying oils of cloves or cinnamon into the colon for their antiseptic, analgesic, and vasomotor effect.

He recommends distensible bags for massage of the atonic sigmoid and rectum, which I hardly advocate.

The gyromele, already described, Türck recommends in the rectum and sigmoid for cleansing the mucosa and producing vibratory movements. I question the possibility of its entering into the sigmoid flexure except very rarely.

Massage, Gymnastics, and Exercise.—The general methods of massage have been described. The course of the colon should be followed. The cannon-ball and vibratory massage are of service.

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[1] Journal American Medical Association, May, 1895.

The portable Vedee vibrator is a useful instrument for the general practitioner. It is illustrated in Part II of this volume. Gymnastic exercises and out-of-door sports are valuable for strengthening the abdominal and intestinal musculature, especially in intestinal atony with constipation. Rowing with a sliding seat, golf, horseback-riding, bicycling, and walking are useful.

Mechanical Support.—Adhesive strapping (Rose's belt), abdominal supporters, etc., are indicated in enteroptosis, in atonic conditions of the intestines or of the musculature of the abdominal wall, in hernial protrusions, etc.

Hydrotherapy.—The Priessnitz compress, poultices, and the application of heat or cold locally are useful.

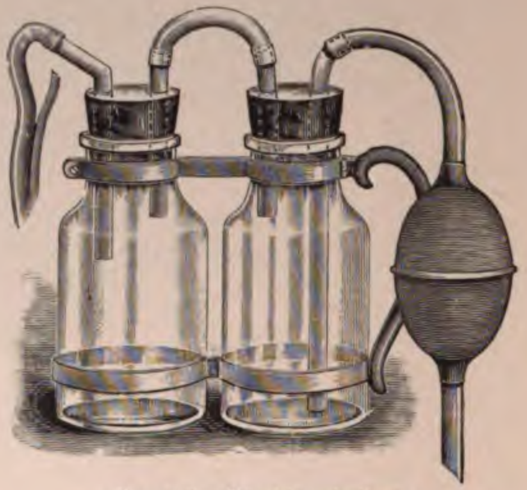

Fig. 198.—Türck's nebulizer.

Sitz-baths and abdominal douches are employed as already described.

Electricity.—Galvanic, faradic, and static electricity may be employed percutaneously. The faradic, chiefly in atonic conditions; the galvanic, in painful neurotic affections. Static electricity can be used for both purposes.

About ten to fifteen minutes is the average period of treatment. When externally applied the current should follow the course of the colon and then be given over the small intestine. Electric vibration may be used.

Violet rays, the high-frequency current, etc., have been advocated for various conditions, but further investigation, I believe, is necessary before they can be recommended. The application of heat and light (electric-light bath) to the abdomen in painful conditions due to a gouty or rheumatic tendency, or in enteroptosis or mucous colic its application to the sensitive areas may prove of service.

Intrarectal Electricity.—Boudet's electrode presents the disadvantage that sometimes frequent evacuations necessitate withdrawal of the electrode.

The author's instrument (Fig. 189) consists of a recurrent irrigator, external tube hard rubber, internal tube metal, with an attachment for a battery pole; a sponge attached to the other pole is placed over the abdomen. Continuous enteroclysis is given with hot normal saline solution, the water carrying the current.

The treatment should be given for five to thirty minutes, depending on the indications. It is excellent for simple atonic constipation and for intestinal paresis. The faradic current is preferable, as strong as the patient can bear. The galvanic may be employed, with negative pole in the rectum, with a current of 10 to 15 milliampères.

CHAPTER XXI

DIET; INTESTINAL DYSPEPSIA; INDICANURIA; SACCHAROBUTYRIC PUTREFACTION; BOTULISM; HYDROGEN SULPHID AUTO-INTOXICATION; ENTEROGENIC CYANOSIS; METEORISM; ENTERALGIA; VISCERAL ARTERIOSCLEROSIS; ANOMALIES; INTESTINAL SAND

DIET

THE general principles of diet were described in Diseases of the Stomach. I will briefly mention a few general rules which should be applied.

Acute intestinal disorders must be managed on the principle of rest. Light food (chiefly liquid) should be given, such as broths, barley-water, gruels, kumyss, matzoon, bacillac, lactone-buttermilk, and in some cases milk, but diluted with barley-water or lime-water. It has been demonstrated that in the acute intestinal catarrh of infants and children, undiluted (or at times even diluted) milk will frequently pass undigested and intensify the inflammatory process, so that reliance is placed for a time on barley-water and similar preparations.

Seibert has demonstrated conclusively that milk is not the ideal food for typhoid fever.

In chronic intestinal disease, for a brief period, rest may be given to the intestines by means of a fluid diet, but feeding should soon be increased, the general nutrition improved, and the intestional tract gradually accustomed to a regular diet, if such be possible. Milk, cream, raw eggs, carbohydrates, and fats are of value to improve nutrition.

In these chronic cases especially, and also in intestinal dyspepsia, all or some special digestive function may be disturbed, such as for proteids, fats, or carbohydrates. The presence of indicanuria, the test diet with stool examination, the fermentation test, together with the clinical symptoms, will determine the type of disturbance of the intestinal digestive function. *The findings would modify the diet.*

Dietetic measures are of special value in reference to disturbances accompanied by constipation or diarrhea.

1. *Laxative foods* are articles which increase intestinal peristalsis. Among such are fruit juices and raw and cooked fruits, such as apples, pears, plums, peaches, strawberries, gooseberries, dates, and figs. Salads, garden vegetables, due to water contained and indigestible residue, such as melons, cucumbers, spinach, tomatoes, pumpkins, and cabbage; cider, bonny-clabber, kumyss, matzoon, and bacillac,

through formation of acid products and gas, increase peristalsis. Some of the fruits and vegetables mentioned have a similar effect. Brown bread and oatmeal tend to move the bowels, also water or carbonated waters. Sugar has a laxative effect. Considerable fat, in the form of cream, butter, or olive oil, aids bowel action.

2. *Constipating Foods.*—Among such are substances containing astringent agents, especially tannic acid, such as tea, dried bilberries, French red wines, cocoa, the acorn preparations, such as acorn coffee, acorn cocoa; mucilaginous foods, such as sago, tapioca, rice, and barley; also foods which leave little residue and exert no irritation, such as albumen-water (white of egg dissolved in water), scraped raw beef, mutton broth, etc.

Pathologically, excess of carbohydrates may be a cause of diarrhea, as may *excess of proteids.*

Milk is constipating in one person, laxative in another, and has no special effect on others. Boiled milk is usually constipating.

Dietetics have changed markedly during the last few years. In chronic colitis with diarrhea the chief diet was formerly scraped meat. Modern methods allow a much greater variety, and the employment of mashed potatoes and boiled rice lessen peristalsis and are often of considerable value.

Certain foods, when taken under ordinary conditions, have no marked influence in increasing peristalsis; among these are meats, fish, meat powders, artificial foods, such as peptone, nutrose, somatose, sanatogen, plain or flavored; eggs, well-baked bread (wheat), crackers, zwieback, and butter or fat in small amount.

Marked seasoning of foods increases peristalsis. The finer the particles of food, the less the irritation; the coarser they are, the greater stimulation they produce on the intestines.

Gelatin.—I have referred to the value of gelatin in ulcer of the stomach and in hyperchlorhydria. Gelatin does not build up new tissue, no matter how much is ingested, though it may diminish tissue waste (Voit).[1] It cannot be reconverted into a proteid.

Kirchmann[2] shows that gelatin *spares proteid in metabolism.*

The ingestion of 7.5 per cent. of the total heat[3] requirement of the organism in the form of gelatin spares 23 per cent. of the body's proteid, while 60 per cent. gelatin reduces it 35 per cent. The small amount of gelatin has nearly as much effect as larger quantities. Its value in typhoid is, therefore, evident to lessen *nitrogen excretion.*

Kaufmann[4] shows that when the lacking tyrosin, cystein, and tryptophan are mixed with gelatin in the proportion in which they occur in true proteid, and are given to a dog or man, nitrogen equilibrium may be established.

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[1] Hermann's Handbuch Stoffwechsel, 1881, p. 396.

[2] Zeitschrift für Biologie, 1900, Bd. xl, p. 54.

[3] One gm. of gelatin furnishes 4.1 calories. About 50 gm. of gelatin represents this requirement in a person weighing 154 pounds.

[4] Pflüger's Archiv., 1905, Bd. clx, p. 440.

Gelatin does not yield indol, and can be employed in the form of jellies as a nitrogenous substance to replace proteids in cases of indicanuria (Herter).[1]

In severe inflammation of the intestines artificial feeding may be necessary, by rectum or subcutaneously.

Rectal alimentation has been described under Diet, in Part II.

Sterile olive oil, ℥j to ij (30.0–60.0), may be injected subcutaneously two or three times a day between the crest of the ileum and lower border of the ribs (preferably). Sterile almond oil in doses of ʒj to ij (4.0–8.0) may be substituted. I do not advocate, however, the subcutaneous method.

Normal saline solution, Oj (500 cc.) to 1 quart (liter), may be slowly injected in the same region in case of collapse or hemorrhage.

INTESTINAL DYSPEPSIA

With intestinal catarrh or biliary or pancreatic obstruction there is perversion of intestinal digestion. These conditions will not be considered here.

Digestive processes in the intestine may become abnormal when the intestinal contents are no longer normal. This may occur even with *intact mucosa and with sufficient bile and pancreatic juice.* Indigestible food, or excess in some special types of food, may lead to fermentative or putrefactive processes in which bacteria take part, and may produce dyspeptic symptoms.

Indicanuria or saccharobutyric putrefaction occur from these conditions.

There may be functional disturbances of pancreatic digestion for the proteids, carbohydrates, and fats, or any one or two of these functions may be disturbed.

Riegel describes cases with marked fermentation of the carbohydrates. There is a jejunal diarrhea of gelatinous semifluid character, often quite green, with bile-pigment reaction and acidity. There is mucus in these movements, but no formed elements, such as epithelial and round cells, which occur in catarrhal conditions. Catarrh may ultimately result.

Herter[2] shows that there is an intestinal indigestion in children of marantic type, in which there is intolerance to carbohydrates, and light colored and gray or fatty stools occur. Indican and phenol are found in large amount in the urine. A great number of Gram-positive bacilli of the Bacillus bifidus type occur in these cases.

Schmidt and Strassburger describe "intestinal fermentation" as dyspepsia in which the stools are light yellow, foamy, with the odor of butyric acid. Fatigue, discomfort, loose stools, or even diarrhea may occur. Abdominal pains, distention, and some tenderness may be present. These cases correspond to saccharobutyric putrefaction (Herter).[1] Dyspeptic symptoms may be due to the decomposition of proteids.[2] Various symptoms, as stated, may be present with intestinal dyspepsia, such as distention, pain, borborygmi, passage of flatus, feeling of discomfort in the abdomen. Diarrhea or irregularity of the bowels is present; loss of appetite, eructations, nausea, and even vomiting may occur. The best method of determining the intestinal functions is by the Schmidt *test diet*, with *examination of the stool.*

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[1] Bacterial Infections of the Digestive Tract, p. 267.

[2] Ibid., p. 285.

Treatment.—That variety of food must be limited for which the particular disturbance exists. Liquid or semiliquid diet may be necessary. *Indicanuria* and *saccharobutyric putrefaction* must be *appropriately treated.*

Lactic acid has been recommended in the form of fermented milks, such as kumyss, matzoon, bacillac, kefir, or lactone-buttermilk; chicken, jellies, gruels, etc., can be given later.

Calomel, gr. $\frac{1}{40}$ (0.0016) four times a day, is suggested by Van Valzah[3] for fermentation; or resorcin, gr. 5 (0.3) t. i. d., is excellent.

Taka-diastase, gr. 5 (0.3) t. i. d., is valuable in amylaceous dyspepsia[4]; or pancreon, gr. 5 (0.3) t. i. d., in disturbances of fat and proteid digestion,[5] and pancreatin, same dosage.

In conclusion, I would advise that in the milder cases the special food stuff which causes dyspepsia should be limited in quantity, but not entirely cut off. Eggs, soft boiled or raw, vegetables in mashed forms, bread or crackers, stale and well broken up, and sanatogen are of service; butter or cream according to indication. Later the diet is increased.

INDICANURIA

Indicanuria designates the presence of indican in the urine, as demonstrated by reactions with the formation of indigo, on using Jaffe's, Obermeyer's, or similar tests.

Indol is absorbed from the intestines and forms in the liver indoxyl potassium sulphate or indican, an ethereal sulphate which is eliminated in the urine.

There are various causes of indicanuria: excessive proteid diet, catarrh of the small intestine, causing alterations in the mucosa and increased intestinal putrefaction, typhoid, cholera, etc., constipation, alimentary putrefaction, partial or complete obstruction of the common bile-duct, decrease of normal digestive fluids, intestinal obstruction, and peritonitis. Certain drugs, such as salol, salophen, and creosote, give reactions which must not be mistaken for it, while urotropin causes its disappearance.

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[1] Bacterial Infections of the Digestive Tract, pp. 294, 300. [2] Ibid., pp. 280, 306.

[3] Medical News, Jan. 17, 1903.

[4] Cellasin, gr. 5 to 10 (0.3–0.6), given t. i. d. an hour after meals in combination with bicarbonate of soda, gr. 10 (0.6), has of late proved valuable in amylaceous and fatty dyspepsia.

[5] True Intestinal Dyspepsia (Einhorn), American Jour. of Med. Sciences, November, 1907; also Med. Record, September 4, 1909.

Intestinal indicanuria is an evidence of intestinal putrefaction. Excessive quantity of proteid, especially of meat, may be a cause. Any condition favoring stagnation in the intestines helps produce this condition. Imperfect action of a cathartic can produce indicanuria.

In children little indican appears. Many adults show indican and suffer from no symptoms, but this is true of constipation. Finally, persons with indicanuria show clinical evidences of intestinal disorder and sometimes symptoms of auto-intoxication, frequently affecting the nervous system. Neurasthenic and even melancholic symptoms may be dependent on this form of auto-intoxication.[1] Intestinal putrefaction[2] may directly affect the liver and kidneys. I have a patient with marked indicanuria with *bile in the urine*, albumin casts, cylindroids, and diminished urea. Treatment of the intestinal putrefaction cleared up this condition.

This corroborates the view of W. H. Porter[3] that excessive intestinal putrefaction may cause disturbance of the hepatic cells. There was no jaundice in this patient. The colon bacilli, through their activity in the decomposition of proteids, are chiefly responsible for the production of indol. The Bacillus aërogenes capsulatus and the Bacillus putrificus, when present, favor the putrefactive action of the colon bacilli on the proteids.[4]

The following test is reliable: Place in a test-tube equal quantities (10 cc. of each) of urine and chemically pure concentrated hydrochloric acid. To this mixture add 3 drops of ½ per cent. solution of potassium permanganate. Then add a small portion of chloroform, 1 or 2 more drops of the permanganate solution, and a few drops more of chloroform, or a total of 75 drops (5 cc.) of chloroform, and shake vigorously for a few seconds.

Indigo-blue is deposited in presence of this indicator. Bile should be tested for even if there are no evidences of jaundice. W. H. Porter's scale (Plate I) is a basis of comparison in the absence of bile test. The color scale of complex indicanuria is omitted.

Rosenbach's test, which consists in boiling the urine with nitric acid, gives a Burgundy red if putrefaction is present. This may occur when no indican is found. It is due to substances of a like class. Taylor[5] recently reports transient heart block due to indicanuria.

Treatment.—*Diet.*—Matzoon, kumyss, lactone-buttermilk, bacillac; later, stale crackers with butter, boiled rice, and jellies (gelatin) are to be added. Herter shows that clinically the carbohydrates have an influence, and the substitution of a quickly digested carbohydrate, like rice which has been well cooked and forced through a colander,

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[1] Headache, migraine, myasthenia, epileptiform seizures, early fatigue, cyclical vomiting, or progressive muscular atrophy may also be dependent on this condition (Herter).

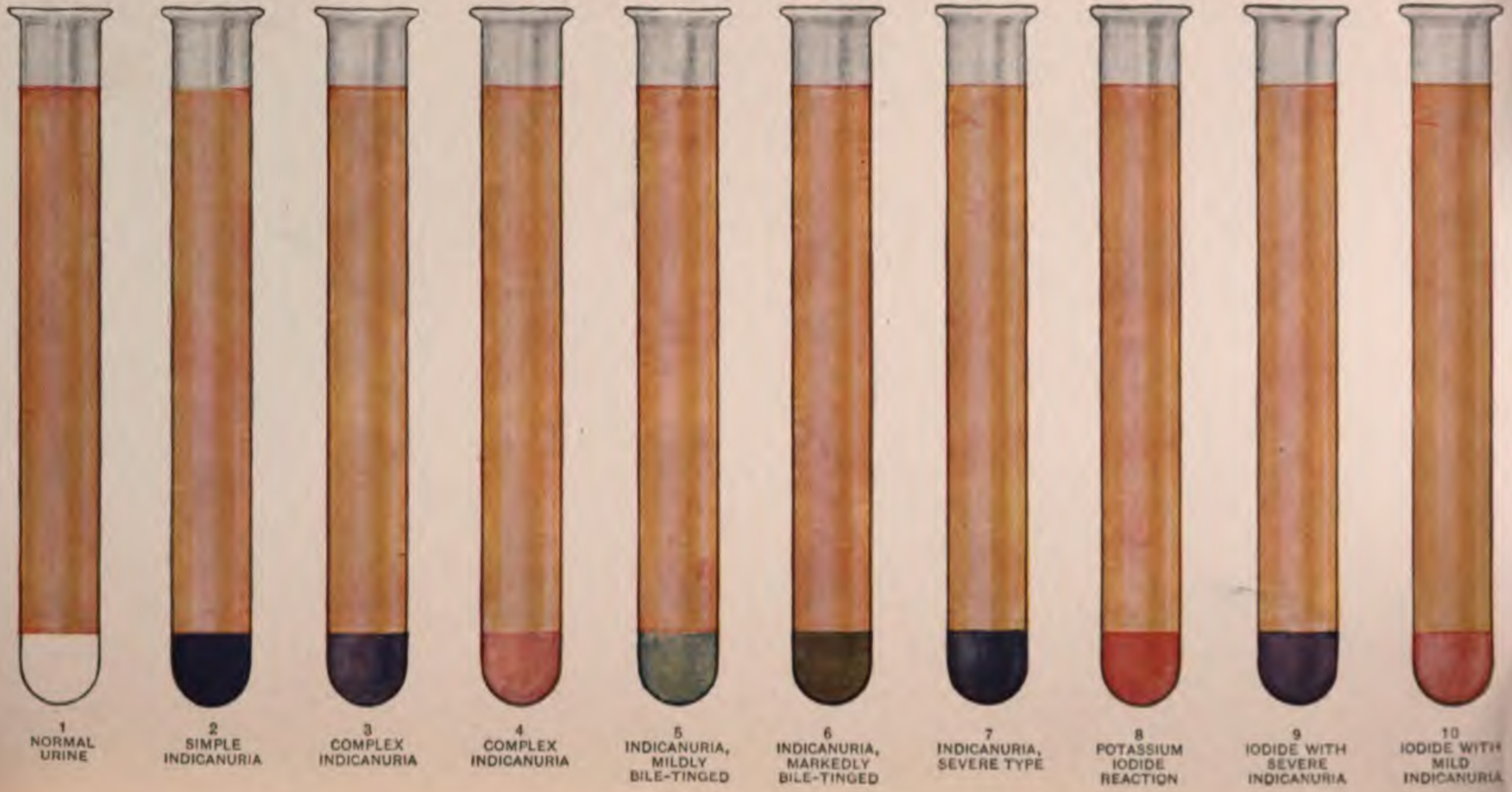
[2] This condition, I believe, further influences the production of arteriosclerosis.

[3] The Post-graduate, Oct., 1902.

[4] Herter, Bacterial Infections of the Digestive Tract.

[5] Jour. Amer. Med. Assoc., April 18, 1908.

PLATE I


1 NORMAL URINE
2 SIMPLE INDICANURIA
3 COMPLEX INDICANURIA
4 COMPLEX INDICANURIA
5 INDICANURIA, MILDLY BILE-TINGED
6 INDICANURIA, MARKEDLY BILE-TINGED
7 INDICANURIA, SEVERE TYPE
8 POTASSIUM IODIDE REACTION
9 IODIDE WITH SEVERE INDICANURIA
10 IODIDE WITH MILD INDICANURIA

INDICAN COLOR SCALE

for large quantities of bread or sugar, will lessen the excretion of indican. Taka-diastase or cellasin can also be given.

Medication.—Blue mass, gr. 5 (0.3); calomel, gr. 5 (0.3), once a week, and a saline (cathartic for a short period) daily.

Urotropin, gr. 5 (0.3), with benzoate of soda, gr. 10 (0.6) t. i. d., has cleared up many cases, albumin casts, bile, and indican disappearing within a short time. This shows the improvement was not due to the mere interference with indican reaction. Lacto-bacillin tablets are useful. Those advocated by Metchnikoff are best.

Aspirin, salicylate of soda, and salol, gr. 5 to 10 (0.3–0.6), are of service, as is also chologestin, ʒij to iij (8.0–12.0), t. i. d.

Enteroclysis is valuable, especially the injection of 1 pint to 1 quart (500–1000 cc.) of a 1 : 1000 acetozone solution, with the patient in the knee-chest posture. This should be retained for a short time. This procedure can be carried out every day or few days. *Gastric disturbances should be investigated and corrected.*

George A. Tuttle has shown that potassium iodid, especially in cases with arteriosclerosis, will lessen indican. Basham's tincture has been of benefit in some. Some have advocated acetozone solution, (1 : 1000), 500 cc. (1 pint), in divided doses daily by mouth.

SACCHAROBUTYRIC TYPE OF INTESTINAL PUTREFACTION

This type of decomposition is produced chiefly by the Bacillus aërogenes capsulatus,[1] Gram-positive. Bacillus putrificus or Gram-positive diplococci may at times be associated.

The Bacillus aërogenes under anaërobic conditions can attack carbohydrates and proteids. Butyric acid is often formed in considerable amount, and often propionic, caproic, or valeric acids. The odor of the movements is often intense and characteristic of butyric or caproic acid. Excessive gas is liberated. The seat of this process is chiefly in the large intestine and lower ileum.

When proteids are attacked there is less gas liberated. With excessive gas production the feces have a low specific gravity and float on water. There are small bubbles in the contents and the feces are a light color. Patients who suffer from this condition do not tolerate well either carbohydrates or acids, flatulence and diarrhea occurring after use of cereals, starchy food, and especially sugar. The mucous membranes of the digestive tract are easily irritated, and there may be epithelial desquamation in the mouth. In the simple cases indol is generally absent.

Combined Saccharobutyric and Indolic Type.—The Bacillus aërogenes capsulatus is also able to break down proteids into a suitable form for the use of other putrefactive bacteria, among which are the indol-forming organisms.

This last form, associated with the excessive production of indol, is the most severe type, and these cases are subject to auto-intoxication, depressive mental conditions, and diminution in muscular power (muscle fatigue), according to Herter.

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[1] Herter, Bacterial Infections of the Digestive Tract.

In the severe types the anaërobic organisms, especially the Bacillus aërogenes capsulatus, produce hemolytic substances which are believed to have a bearing on the production of pernicious anemia.

The results of intestinal irrigation in such cases have been favorably reported by Hollis[1] and Ditman,[2] as in Fig. 199, with marked improvement in the hemoglobin and red cells.

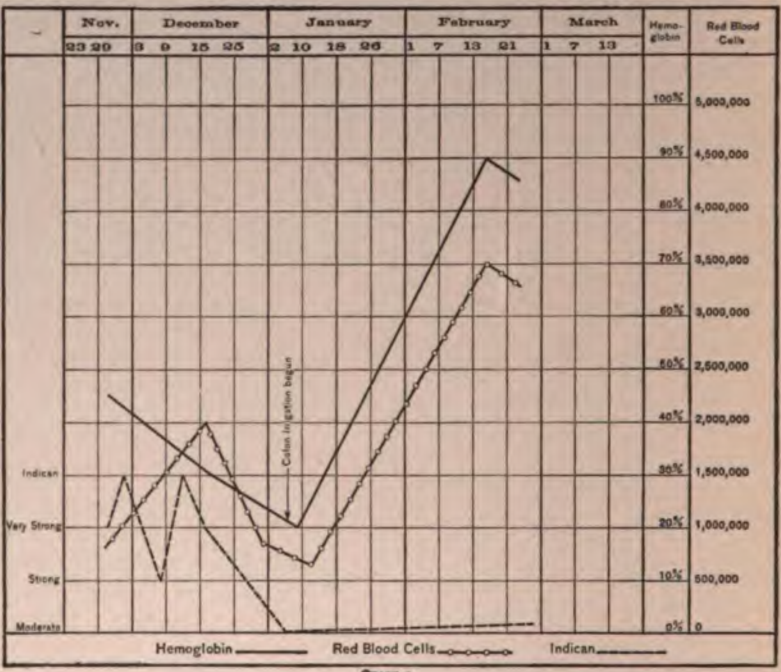

CHART 1.

Fig. 199.—Chart of pernicious anemia demonstrating value of enteroclysis.

Arthritis deformans[3] has recently been imputed to putrefactive changes in the intestinal canal. At the Red Cross Hospital excellent results were secured by the author by enteroclysis and fermented milks, no medication; the swelling in the joints rapidly subsiding.

Treatment.—Careful mastication of the food, cleanliness of the mouth, and correction of gastric disturbances are necessary. Sugars should be omitted, starchy food cut down, and meat diminished or eliminated if the mixed type with indican is present.

Taka-diastase, gr. 5 (0.03) t. i. d., aids starch digestion. Pancreatin preparations may be tried. The fermented milks are of special value. Lactobacillin tablets are useful.

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[1] Herter, Bacterial Infections of the Digestive Tract.

[2] Medical Record, Feb. 2, 1907.

[3] A Preliminary Report on the Relation of Albuminous Putrefaction in the Intestines to Arthritis Deformans by Andrews and Hoke, American Journal of Orthopedic Surgery, July, 1907.

Enteroclysis, or high enema, with saline solution, acetozone (1:1000) or hydrogen peroxid (1:1000) are of service. The latter are of special value.

Ichthoform, formidin, ichthalbin, aspirin, salol, salicylate of soda, gr. 5 (0.3) t. i. d., are useful;[1] or urotropin, gr. 5 (0.3), with benzoate of soda, gr. 10 (0.6), t. i. d. This last combination, with the addition of the high enema of acetozone 1 quart (liter), strength 1:1000, I have found of great value. Surgery[2] may be indicated in extreme cases.

BOTULISM

This is a variety of meat-poisoning due to the Bacillus botulinus, isolated by Van Ermengen from raw ham. The symptoms resembled those of so-called sausage-poisoning. This same type of poisoning has occurred from eating beans that were imperfectly canned. These were probably infected through the manure of pigs. Meat may be extensively infected with the Bacillus botulinus, and contain a large quantity of the toxins, without showing the signs of decomposition. The filtered toxins can produce the effects.

Incubation.—The symptoms seldom appear before twelve to twenty-four hours after ingestion of the infected meat. They are: First, disturbances of the external muscles of the eyeball, such as ptosis, abducens paralysis, disturbances of associated movements with nystagmus; second, disturbances of the internal muscles of the eyes, such as enlargement and rigidity of the pupils; third, there are swelling or paralysis of the tongue, pharyngeal and laryngeal paralyses, and disturbances of the heart and respiration; fourth, there may be weakness or paralysis of motion. Changes in sensibility and consciousness usually do not accompany these disturbances. Fever has frequently been absent and there has even been absence of disturbances of the stomach and intestines. This condition is not so rare abroad and occurs in this country.

Treatment.—Evacuation of the gastro-intestinal tract. Treatment of symptoms. Herter recommends Kempner's antitoxin when procurable.

HYDROGEN SULPHID AUTO-INTOXICATION

In health the formation of hydrogen sulphid seems probably limited to the large intestine and a small adjacent portion of the small intestine. Under pathologic conditions it is formed in other parts of the digestive tract. In chronic ectasia hydrogen sulphid is at times liberated when putrefactive changes take place. Herter believes the eructation generally occurs from this viscus, so that no symptoms result. He holds that the stagnating stomach contents probably go on to the formation of cystin, and that the Bacillus lactis aërogenes, Bacillus coli, and other bacteria produce hydrogen sulphid therefrom. Senator[1] reports a case of auto-intoxication from hydrogen sulphid (hydrothionemia). After an error in diet the patient became ill with gastro-enteric catarrh. On the third day vomiting accompanied with the odor of hydrogen sulphid occurred. The patient became dizzy and went into collapse, without loss of consciousness. The eructation of gas continued, and the first urine gave the reaction of hydrogen sulphid. The case recovered.

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[1] Permanganate of potash up to gr. 1 (0.06) t. i. d. may be of service. Herter suggests the possible value of dioxid of manganese.

[2] If dilatation of the stomach, not responding to medical treatment, is the cause, then gastro-enterostomy is indicated. With failure of medical treatment and progressive anemia and mental disturbances, Herter suggests appendicostomy followed by enteroclysis. I am opposed to shortening or short circuiting the large intestine for this condition. In extreme cases Gant's cecostomy, with irrigation of colon and ileum, might be of service.

Betz[2], and Stefanio and Emminghaus[3] describe cases. Headache, dizziness, delirium, mental depression, drowsiness, stupor, and collapse were prominent symptoms.

Treatment.—Catarrh of the gastro-intestinal tract, ectasia, and intestinal putrefaction must receive appropriate treatment as required.

ENTEROGENIC CYANOSIS

This peculiar type of auto-intoxication was first described by Stokvis.[4] The patient, aged fifty-eight, suffered from severe enteritis with pronounced cyanosis of the skin and visible mucous membranes, together with a swelling of the terminal phalanges. Spectroscopic examination of the skin and mucous membranes showed a band corresponding to the absorption spectrum of methemoglobin. It was believed that poison substances were formed in the intestine which transformed part of the hemoglobin into methemoglobin. Talma[5] reported 3 cases. Van der Bergh[6] reported 2 cases, one of which was evidently due to sulphemoglobin (from hydrogen sulphid) in the blood, and in the other the blood contained nitrites.

Van der Bergh also reports a case in a child nine years of age, who suffered from marked digestive disturbances with diarrhea. Anuria was marked. Child was very cyanotic and the ends of the fingers were clubbed; abdominal distention marked. Urine contained no albumin, no sugar, but a little indol. No cardiac affection present.

Several cases of sulphemoglobinemia have been associated with obstinate constipation, the relief of which has been followed by improvement.

Treatment.—This should be directed to the intestinal tract. Proteids should be reduced; sour milks given; enteroclysis and the general treatment of intestinal putrefaction.

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[1] Berlin. klin. Wochenschr., v, p. 254, 1868.

[2] Memorabilien, lx, p. 145, 1864.

[3] Berlin. klin. Wochenschr., lx, pp. 477, 491, 1872.

[4] Festsch. f. v. Leyden, l, p. 597, 1902.

[5] Tijdschrift voor Geneesk., li, p. 721, 1902.

[6] Deutsch. Archiv. f. klin. Med., xciii, p. 86, 1905; also Berlin. klin. Wochenschr., No. 1, p. 7, 1906.

METEORISM; TYMPANITES

This is defined as an accumulation of gas in the intestines. Flatulence is used to indicate a great formation of gases that are removed by eructations and flatus.

Part of the gas thus accumulated is expelled and part is absorbed, and thus the volume of gas in the intestines is regulated.

Etiology.—The causes of tympanites are as follows:

1. An increased introduction (ingestion) of gas.
2. The development of excessive gas within the intestines.
3. A diminution or impairment of the eliminative power of the intestines for gases.

1. *The increased introduction of gases may be due to the excessive drinking* of aërated beverages or the swallowing of air.

Aërophagy (air swallowing) usually occurs in hysteric women, and may result from shock or emotional disturbance. It is generally involuntary, due to spasm of the pharynx. The symptoms are distention after food, loss of appetite, frequent noisy eructations, often insomnia or sleeplessness, constipation. Frequently mucous colic or gastroptosis (enteroptosis) coexist. Rapid deglutition movements precede the eructations.

The mucous colic or enteroptosis should be treated; the hysteria combated; pharyngeal spasm checked by keeping the mouth widely open, applying cocain (1 per cent.) locally, blisters externally, belladonna, bromids, or valerian internally; hypnotic suggestion in some cases; strychnin to stimulate the muscular tone of stomach; food should be concentrated.

2. *Formation of Abnormal Quantity of Gas in the Intestines.*—This may be due to fermentation of the carbohydrates or to putrefaction of the proteids. An excessive amount of fermentable or putrefactive material or food which cannot be assimilated are factors. Catarrhal conditions influence the activity of the ferment and putrefactive organisms.

3. *Diminished Elimination of Gases from the Intestines.*—This is due to mechanical obstruction or to a reduction or inhibition (paralysis) of the muscular power of the intestinal wall.

Among the causes are stenosis, intestinal obstruction, paresis of the intestines in the infectious diseases, such as typhoid fever, pneumonia, cerebrospinal meningitis, peritonitis, etc.

In pathologic conditions, where there is circulatory disturbance in the intestinal walls, as in peritonitis, etc., absorption of gases must be interfered with. It is difficult to estimate to what degree this is a factor.

Nervous Meteorism.—This is most common in the hysteric, and usually occurs as a diffuse distention of the abdomen (tympanites hystericus), but also as a circumscribed swelling (phantom tumor).

Various factors have been suggested, such as swallowed air;

insufficiency of the pylorus, allowing passage of air from stomach to bowels; or continuous contraction of the diaphragm (Talma).

With narcosis the abdomen becomes flaccid, while with returning consciousness meteorism recurs, and air cannot be detected entering or leaving the tract, hence Talma denies that there is any increase of gas in the intestines. Some attribute the condition to acute general paresis, of sudden onset, passing off rapidly, analogous to other hysteric paralyses.

Symptoms.—The shape of the abdomen is usually altered, a feeling of tension is almost always experienced. With general meteorism the abdomen is distended quite symmetrically, while with partial distention the affected parts stand out in marked relief.

With local distention the coexistence of visible peristalsis, as with stenosis, simplifies the diagnosis. The degree of distention generally corresponds to the amount of gas present; but the weaker the muscles and the more reduced their tone, the greater is distention. An example of this is marked tympanites in peritonitis.

When the abdominal muscles are tense sometimes the diaphragm, heart, and lungs are forced upward, and severe dyspnea results, which occasionally may be followed by a fatal issue.

In severe cases there is a constant feeling of pressure and a desire to pass wind, while colicky pains are sometimes present. As a rule, no flatus is passed, or in inconsiderable amounts at long intervals.

Palpation shows the abdominal walls are very tense. They present an air-cushion resistance. The percussion note is abnormally low and loud, the tympanitic ring is usually lost. With auscultatory percussion a metallic sound is elicited. The liver dulness may be reduced and finally disappear. There are some cases of meteorism, in which the wind is passed almost constantly from the anus with considerable noise. Rosenheim believes, as these gases are odorless, that the air is pumped into the rectum and emitted again as flatus.

Prognosis.—If occlusion of the intestines is present, the outlook is serious. Ordinary cases are favorable.

Treatment.—This should be directed toward removal of the cause responsible for meteorism, such as peritonitis, typhoid, stenosis, etc.

Drinks and foods known to produce flatulence must be prohibited. Avoid carbonated waters, beer, champagne, cider, excessive carbohydrates, rich pastry, etc.

Intestinal fermentation or intestinal putrefaction, if they cause the meteorism, must he treated after the methods already described.

Among the intestinal antiseptics are ichthalbin, ichthoform, formidin, salol, salicylate of soda, benzonaphtol, urotropin, and sodium benzoate, average dose of each, gr. 5 (0.3) t. i. d. after meals. They may be given in shellacked capsules. Bismuth salicylate and bismuth subnitrate, gr. 5 to 10 (0.3–0.6) t. i. d., are of service.

Calcined magnesia, lime-water, and charcoal are suggested to absorb the gas.

In mild forms of flatulency various carminatives have been employed, such as caraway seed, peppermint, mint, thyme, cinnamon, cloves, anise seed, nutmeg, sassafras, and fennel. These are best given as infusions. Asafetida and the oil of cloves are believed by Brunton to aid absorption of CO_2 and H_2S.

Cathartics and laxatives may be necessary when there is no peritonitis or obstruction. Physostigmin salicylate, gr. $\frac{1}{100}$ to $\frac{1}{80}$ (0.0006–0.001), has been recommended in severe cases.

Massage, abdominal douches, electricity, and electric enteroclysis are of service in cases where there are no anatomic lesions. Friction of the abdomen with spiritous, aromatic, or ethereal substances, such as camphorated oil, turpentine, oil of cajeput, etc., is serviceable.

The introduction of a colon-tube may aid the escape of gas. Water enemata of soapsuds, containing spirits of menth. piperit., ʒj (4.0), or oil of turpentine, ʒj (4.0), are useful.

Puncturing the intestines with a trocar is a dangerous procedure.

Meteorism in the hysteric occasionally disappears without treatment. Attention should be paid to the nervous condition. Valerian and asafetida are useful by mouth or enema in such cases.

Pill asafet., one t. i. d.; emulsion (milk) asafet., ℥j to ij (30.0–60.0), by enema.

Zinc valer., gr. 2 (0.125), or ammon. valer., gr. 5 (0.3), t. i. d.

Tinct. valer. with tinct. lavend. co., *āā* ℥ij (60.0). Dose, ʒj (4.0) in water t. i. d. after food. Iron, arsenic, and strychnin are of tonic value in the nervous cases.

Massage, friction, and a tight abdominal band are useful.

INTESTINAL PAIN (INTESTINAL COLIC; ENTERALGIA)

Intestinal pain can be distinguished as follows:

1. Pain originating from inflammation of the intestinal wall or of its peritoneal coat.
2. Pain of colic.
3. Nervous enteralgia, described under Neuroses.

Colic is the painful stimulation of the intestinal nerves which is produced by severe tonic contractions of the intestines. G. F. Shiels[1] holds that the pain is produced in the peritoneal coat. Kast and Meltzer have demonstrated by a series of experiments that the intestines are sensitive to pain.

Etiology.—Organic lesions of the intestines, excess in or improper articles of food, cold drinks, substances causing marked gas formations, fecal accumulation, intestinal worms, foreign bodies, gall-stones, enteroliths, tainted foods, large quantities of mucus, as in mucous colic, exposure to cold, occasionally ulcers, internal strangulation of the bowels, stenosis, purgatives, lead- and copper-poisoning may produce colic.

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[1] American Journal of Surgery, April, 1908.

Symptoms.—The pain of colic is peculiar—pinching, boring, or occasionally of a tearing character—it occurs in paroxysms which may last a few seconds or be prolonged several hours. It usually appears suddenly and disappears as rapidly. Its intensity may be so severe that fainting and collapse occur.

If colic begins from error in diet, there may be gastric disturbances, belching, nausea, and vomiting. Obstinate constipation and flatulence may be present, or if the cause be from improper food, diarrhea. The pain frequently starts at the umbilicus and remains localized or radiates in other directions. The face of the patient shows his suffering. Pressure over the abdomen relieves the pain in some, while in others it increases it. There may be straining sensations in the bladder and rectum and occasionally borborygmi can be heard. Peristaltic movements can be seen in thin patients. If accumulated fecal masses and gas are evacuated spontaneously or by injections, the attack rapidly subsides.

Spastic contractions of the intestines may be encountered. If they involve a large part of the bowels, as in lead-colic, the abdomen appears trough shaped. In stercoral and wind-colic, it is usually tympanitic.

Diagnosis.—In enteralgia due to anatomic lesions of the intestines the pain is increased by pressure. There is frequently diarrhea and the stools contain blood, mucus, and, rarely, pus.

With rheumatism of the abdominal muscles the pain is superficial and not within the abdominal cavity. It often changes its location. Pressure increases the pain, while rest and antirheumatic medicines diminish it.

With peritonitis there are fever, tenderness on pressure, muscular rigidity, meteorism, absence of abdominal respiration, increase of polynuclears, and leukocytosis.

Hyperesthesia of the abdominal wall usually occurs in hysteria and neurasthenia. The pains are superficial, lying chiefly in the skin. The faradic current often removes the pain. Biliary and renal colic are recognized by location of the pain and characteristic symptoms. Neuralgic pains are superficial and radiate.

Prognosis.—These cases end in recovery, with rare exceptions.

Treatment.—For the relief of pain, morphin by hypodermic, gr. 1/8 to 1/4 (0.008–0.016), or codein, same or even double dose, or tincture of opium, ♏x (0.59 cc.), heat to the abdomen, and hot saline enema or enteroclysis at 115° F. Squibb's mixture is useful.

Later, the bowels should be thoroughly evacuated by enemata[1] which may contain 1/2 pint (250 cc.) of olive oil and ʒj (4.0) of spirits of turpentine, and a cathartic—castor oil, ℥iss (45.0), or calomel, gr. 5 (0.3)—be given, followed by a saline cathartic.

The cause of the colic should be corrected, as should any errors in diet. Fluid diet should be employed.

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[1] The simple soapsuds enema, 1 quart (liter).

VISCERAL ARTERIOSCLEROSIS

Harlow Brooks[1] calls to our attention that the presence of diminished visceral function, with occasional and otherwise unaccountable elevations of the blood-pressure, should, particularly in connection with possible etiologic factors, lead to a suspicion of some localized, if not general, area of arterial disease. When associated with pain of peculiar anginal character, location in some special organ may be within the range of possibility. I feel quite certain that careful observation of cases of obscure abdominal pain, paroxysmal in character and associated with elevation of the blood-pressure, will eventually make it possible to diagnose these generally unrecognized types of arterial disease while in a stage when something may still be done for their alleviation or for the prevention of their further progress.

Some of these patients give a history of nervousness, gastric catarrh, and pain; vomiting and even hematemesis may occur. Meteorism and constipation are not uncommon. Pain over the pancreas and intestinal functional disturbances have been noted. When such symptoms occur in a patient with well-marked arteriosclerosis, and are relieved by iodids and nitrates, one is justified in diagnosing abdominal arteriosclerosis.

Etiology.—Alcohol, nephritis, syphilis, old age, tuberculosis, tobacco excesses, and toxemia are the chief causes.

Perutz[2] shows we must differentiate between angina pectoris and angina abdominis due to arteriosclerosis:

I have recently seen several cases who have complained of pain in the epigastrium, belching, constipation, and headache. The gastric analysis showed deficient hydrochloric acid secretion and the pulse the characteristics of arteriosclerosis. Treatment afforded no relief until the nitrites were administered. "Gilbride[3] reports a number of cases with symptoms of pain in the epigastric or umbilical regions, rarely in the lower abdomen. This is increased by exertion and in some cases during digestion. Weakness, loss of weight, abdominal distention, and belching are present. Bowels are constipated or there is constipation alternating with diarrhea, or occasionally normal movements. There may be vertigo or disturbances of vision. The gastric analysis showed in most cases deficiency of hydrochloric acid; in one it was nearly absent and in one normal. Stomach motility normal or increased. Lactic acid and Boas-Oppler bacilli were absent. Cancer had been suspected. The radial pulse and blood-pressure may show arteriosclerosis. In some cases there are no evidences of this, but there are two symptoms significant of abdominal arteriosclerosis—*tenderness of the abdominal aorta* with *epigastric pulsation.* Some patients may have attacks of angina pectoris with pain referred to the epigastrium. The age of the patient is usually over forty." Ortner[1] has contributed to the subject, demonstrating the disturbances of the motor, secretory, and absorptive functions of the intestines due to arteriosclerosis; and Akin[2] has reported several cases. Nitrites afford the most relief, with the addition of potassium or sodium iodid.

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[1] American Journal of the Medical Sciences.

[2] Münch. med. Wochenschr., May 28 and June 4, 1907.

[3] Gastro-intestinal Disturbances Due to Arteriosclerosis, Journal Amer. Med. Assoc., March 20, 1909.

ANOMALIES IN THE POSITION AND FORM OF THE INTESTINES; ENTEROPTOSIS

Various anomalies in the position and form of the intestines occur; in some cases congenital, and in others acquired, of which Koch and Curshman have made a study.

They may be congenital in some cases, acquired through coprostasis, the weight of a tumor, or the formation of adhesions, in the majority of cases the colon being affected. Various angulations of the sigmoid, especially due to adhesions, are described by J. P. Tuttle. Inflation with air and the use of bismuth injections with the *x*-rays aid diagnosis.

Hirschsprung's disease, or congenital primary dilatation and hypertrophy of the colon, is found at birth. Dyspeptic symptoms, obstinate constipation, and colicky pains with great distention are present. By insertion of a finger or catheter into the rectum a movement will occur with relief of the distention. Enteroclysis, electricity, etc., are of value.

Idiopathic dilatation of the colon is a rare event, and enteroptosis, the most common displacement, is described under Gastroptosis.

Idiopathic dilatation of the colon, in which there is no mechanical obstruction to the feces, may be congenital, develop early or later after birth, or be acquired in adult life. Nervous derangements, habitual constipation, rickets, or chronic colitis may be factors. The colon and especially the sigmoid are greatly distended, so that the protrusion of the abdomen may justify the term "balloon man." Constipation or irregularity of the bowels, gaseous distention, intestinal putrefaction, shortness of breath, etc., occur. Enteroclysis, regulation of the bowels, diet, and treatment of indicanuria are indicated. Abdominal support is of service. Rarely, operative procedure is required.

INTESTINAL SAND

This material is gritty, contains organic matter, inorganic salts, especially calcium phosphate and carbonate, but *no cholesterin,* which distinguishes it from biliary sand.

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[1] Volkmann, Samml. klin. Vortr., No. 347.

[2] Jour. Amer. Med. Assoc., June 5, 1909.

It has been considered by some as a manifestation of the arthritic diathesis. It is usually associated with mucous colic or possibly constipation. It occurs generally in women between thirty and forty years of age.

The associated condition must be treated. Sodium bicarbonate and bismuth salicylate have been recommended.

False intestinal sand, such as the residue of vegetable food, especially from *pears*, must not be mistaken for true sand. Symptoms are absent.

CHAPTER XXII

CONSTIPATION AND DIARRHEA

CONSTIPATION

(*Synonyms.*—Obstipatio Alvi; Constipatio Alvi; Atony of the Bowel; Habitual Constipation.)

HEALTHY persons usually have one bowel movement daily, generally about the same hour. Some normally have two actions a day, while others empty the bowels every other or every second day, and yet are in perfect health.

Constipation is defined as a condition in which the feces are not passed sufficiently often. Another form is that in which defecation occurs daily, but the movements are insufficient in quantity. A stagnation of fecal matter may thus occur. The quantity of feces is somewhat variable, from 100 to 150 gm., the average, even up to 250 gm., being greater after a vegetable diet and less after meats.

Considerable of the evacuation is made up of micro-organisms, of which Herter[1] has estimated the daily number as 126,000,000,000, which explains the fact that patients who eat little may pass considerable material.

Constipation may be acute or chronic. The acute type is due to complete obstruction of the intestinal tract or to dynamic ileus (intestinal paresis).

The chronic type is extremely common. Henry Illoway and Samuel Gant have written excellent works on this subject. My classification is slightly modified from their books.

Etiology.—All possible factors must be carefully investigated.

1. *Diseases of the stomach*, such as hyperchlorhydria, ulcer, cancer, dilatation, simple atony, catarrhal conditions, and achylia gastrica may cause constipation.

2. *Obstruction of the bowel* by tumors of the intestine or tumors pressing on the bowel from some adjacent organ; by *stricture within the intestine*; by external stricture, as by peritonitic adhesions; by chronic intussusception.

3. *Catarrh of the small intestine alone;* in some cases catarrh of the large intestine; mucous colic; atrophy after catarrh; ulcers of the small intestine are occasionally attended by constipation; *dysenteric ulcers at times produce constipation, though ulcers of the large intestine usually cause diarrhea.*

4. *Voluntary abstention from stool* on account of the pain it produces, by reason of disease of the rectum, such as from piles, fissure,

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[1] Bacterial Infections of the Digestive Tract.

or ulcer. Increased contraction of the sphincter ani also occurs from irritation and interferes with bowel action.

5. *Obstruction to the entrance of bile into the intestine* or deficiency of bile.

6. *Diseases of the heart, lungs, liver, and kidneys.* Intestinal hyperemia and congestion of the portal system are factors in these cases in retarding peristalsis, as in cirrhosis of the liver.

7. *Disease of the pancreas.*

8. *Diabetes*, anemia, and chlorosis.

9. In *many diseases of the brain, spinal cord, and of the nervous system* constipation is present.

Among such are chronic insanity, diphtheritic paralysis, tabes, brain tumors, cerebrospinal meningitis, hemorrhage of the brain, chronic hydrocephalus, myelitis, neuroses, and psychoses.

10. *Acute febrile conditions are usually accompanied by constipation*, as pneumonia, etc. This generally excludes those with special intestinal lesions. We must remember that constipation occurs in some cases of typhoid, and even of dysentery.

11. *Chronic constipation from foreign bodies.*

12. *Malformations*, such as abnormally developed colon; undue size or length of the sigmoid flexure; diverticula of the large intestine; a diaphragm partially closing the large intestine.

13. *Defective development or essential primary atrophy of the colon.*

14. *Enteroptosis;* angulations of the sigmoid flexure due to adhesions; prolapse of sigmoid into rectum from long mesentery—in effect, slight intussusception.

15. *Atrophy of the intestinal musculature* following catarrh or fatty degeneration, as in consumption or in alcoholics.

16. *Hypertrophy of Houston's valves.*

17. *Loss of power in the abdominal muscles* may be a factor in some cases, as in the emaciated, with multiparous women with diastasis of the recti, etc.

18. Chronic constipation *from impaired physiologic function.* This type is due to disturbance of the motor function of the intestines, and is strictly classified under *motor neuroses*, under which we have:

(*a*) Constipation due to retarded intestinal peristalsis (atony or relaxation of the bowel).

(*b*) Spastic constipation, perverted action, or enterospasm. The constipation is due to a spasmodic contraction of a portion of the intestine.

(*c*) Spasm of the sphincter is included under this type.

Constipation Due to Disturbances of the Motor Function.—Habitual constipation due to impairment of the physiologic function (*i. e.*, caused by motor disturbances) constitutes an important class of cases. It should be strictly classified under *motor neuroses.*

There are the two types mentioned above: the atonic and the spastic forms of constipation.

Atonic Constipation.—Constipation due to atony (literally, relaxation) *of the bowels* constitutes the majority of cases.

Predisposing Causes.—The character and quantity of food and the amount of physical exercise influence bowel evacuation. Boas has laid stress upon the importance of the type of nourishment and its influence in the production of constipation (alimentary constipation).

Albuminous diet consisting of meat and eggs leaves little residue, and eventually tends to constipate; while with vegetable diet there is more residue and the fermentation products excite peristalsis. A patient may diet by avoiding vegetables, butter, and fat, and thus constipation result which may become chronic.

Repeated neglect of the call of nature is a frequent cause, such as in the case of young girls and children while at school, or among women at social gatherings, etc.

Interruption of the habit of regularity, as on a railroad journey, or an attack of diarrhea with subsequent constipation, or of acute fever, may lead to habitual constipation. The same is true of mental conditions, such as depressing emotions. Prolonged exercise, such as long marches, has produced chronic constipation.

Constipation is more frequently found among the wealthy than among the working class. The mode of living, sedentary life, etc., have a bearing. Worry and mental strain have an influence, and it occurs among neurasthenics and hypochondriacs.

Habitual constipation is found in those engaged in active exercise and who are of strong constitution. In such persons the cause of this perversion of motor function is unknown, but it seems inherent to the patients that the bowels respond slowly to stimuli. In some cases there seems to be hereditary or congenital diminution of intestinal peristalsis (constipation).

Riegel observed cases in which at autopsy the musculature of the large intestine was abnormally thin and was probably congenital. In some of these cases the skeletal muscles were strong. Nothnagel described similar cases in which the general muscular development was poor. These rare conditions cannot be recognized *intra vitam.*

Enteroptosis is given as a cause of chronic constipation on account of the intestinal angulation which occurs. This is true in some cases. The general atony[1] with enteroptosis is the chief factor. The musculature of the stomach, intestines, and also the abdominal muscles are relaxed, which have a bearing on the production of constipation. The abuse of cathartics, and repeated distention of the bowel from large enemata, may produce constipation.

Spastic Constipation.—Spasmodic constipation of the bowels, or enterospasm, is produced by a perversion of the motor function of the intestine, taking the form of a spasmodic contraction of a portion of the intestines, which may involve both the circular and longitudinal muscles.

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[1] Atonia Gastrica, Rose and Kemp.

This spastic condition may be of variable duration and involve different segments of the intestines. Fleiner holds that the contracted portion retains its contents, while others believe that it is nearly occluded by the spasm, thus creating an obstacle to the passage of the intestinal contents.

Diffuse enterospasm involving the small intestine occurs in spinal meningitis, in diseases of the pons and medulla, and in chronic lead-poisoning. In these cases the abdomen is retracted like a trough.

Local or circumscribed enterospasm is more frequent, and it generally affects a portion of the large intestine. The abdomen shows no abnormality on inspection.

Spastic constipation is more frequently met with in nervous persons, neurasthenics, the hysteric, and in those debilitated by long-continued disease. The constipation is quite obstinate, lasting for several days. The evacuation is somewhat painful and consists of small balls (goat feces) or pencil-shaped fecal material. There are spasmodic pains in the left lower abdomen or in the umbilical region which are relieved by the passages.

Spasm of the Sphincter (*Proctospasmus*).—This condition really belongs to spastic constipation. There is painful spasmodic contraction of the sphincter. I have seen cases, however, in which pain was not marked, but spasm occurred when defecation was attempted; in effect, spasmodic stricture was present. Such cases may be a direct factor in the production of constipation.

Many cases of sphincteric spasm are secondary to fissure or ulcer of the rectum, or are reflex when there are inflammatory conditions in the neighboring organs, such as the uterus, bladder, etc. Some cases occur as a primary nervous affection and are chiefly met with in those with a nervous taint or disease of the spinal cord.

Rectal examination is often extremely painful. In some cases there is simply reflex spasm on examination, and the sphincter is found to be extremely tight and evidently hypertrophied from frequent contraction.

It is important to consider the possibility of the last type in connection with chronic constipation.

Symptoms.—In many cases constipation causes no subjective symptoms. Some have a stool every second or third day or even once a week. There is a classical case *who had an annual movement.* Nevertheless, chronic constipation should not be made light of.

Some patients on the appearance of constipation immediately suffer from subjective symptoms, which may become at times quite severe. Among milder symptoms are sensations of fulness, tension, and discomfort in the abdomen; at times they are referred to the stomach; loss of appetite, occasionally belching, nausea, pyrosis, and a disagreeable taste in the mouth. Coated tongue and headache are often present. Colicky pains and distention may occur.

These symptoms disappear after thorough evacuation of the bowels.

On inspection and palpation considerable fecal accumulation can be found in the intestines, especially in the colon. Some patients suffer from severe headache, dizziness, sleeplessness, despondency, palpitation, tachycardia, or irregular pulse. Such symptoms I believe due to auto-intoxication from the intestinal tract. *Motor insufficiency, as in constipation, favors stagnation and putrefactive changes.*

Duprey[1] reports extreme cases in which the patients became unconscious, but recovered after free bowel evacuation. One death also occurred.

In most of my patients suffering from chronic constipation, with the symptoms just described, especially with nervous manifestations, marked indicanuria was present.

Herter[2] states that most children and many adults may fail to develop indicanuria with constipation, yet there are others in whom it is marked, and that a *satisfactory explanation is not possible.*

In my report of 13 cases of dementia paralytica before the American Medicopsychologic Association[3] marked constipation was noted in all the cases, and all suffered with considerable temperature. Rectal irrigation lowered the temperature in 3 cases, and under general treatment for the gastro-intestinal tract it was lowered in 8 more. Convulsions diminished in 5 cases. The bowels were freely opened.

Bouchard's theory that no intoxication from the intestinal tract can take place when the feces are solid, I believe untenable. Dunin has shown that constipation may be the result of nervous conditions. There are many in which no nervous factor can be discovered, and others in whom constipation and intestinal auto-intoxication are factors in the production of nervous symptoms.

Termination.—The bowels may act spontaneously, hard masses of fecal matter being passed covered with a thin layer of mucus. Feces are often passed in small balls or in rod shape. With the atonic type of constipation relief is usually felt after defecation; while with the spastic type movement is accomplished with great effort, and there is a feeling as if there were still material in the rectum.

In some cases the constipation terminates in an attack of diarrhea due to hyperemia and the secretion of fluid from irritation of the mucosa by the hardened feces. In others, purgatives or enemata may be required, or removal of scybalæ from the rectum by the fingers.

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[1] Lancet, 1902, p. 1832.

[2] Bacterial Infections of the Digestive Tract, p. 263.

[3] Proceedings of the Sixty-first Annual Meeting, April, 1903. Some observations on the Relations of the Gastro-intestinal Tract to Nervous and Mental Diseases.

Slight catarrh may occur or, rarely, stercoral ulcers, local peritonitis, or even perforation and general peritonitis. Constipation has been a factor in the production of typhlitis, diverticulitis, catarrh of the cecum with secondary catarrhal appendicitis, volvulus, and subacute or acute intestinal obstruction.

Fecal Colic.—When large masses of fecal matter accumulate, colic may occur. The patient is seized with violent colicky pains which may cause a fainting spell. The abdomen is distended and tender. Passage of flatus brings temporary relief. The symptoms do not disappear until thorough evacuation of the fecal accumulation takes place. Fecal colic may occur, with daily evacuation of the bowels. The detection of fecal accumulation by palpation is of chief importance.

In obstinate cases of constipation cathartics may fail to produce movements and the patient go into marked collapse and vomit profusely, with symptoms resembling intestinal obstruction. Rectal irrigations or oil injections may relieve the condition.

Rarely, in the insane, old, or weak total paralysis of the colon may take place and the patient die with the symptoms of obstruction. In cases of fecal accumulation it is always safer to employ injections and irrigations before resorting to active catharsis.

Fecal Tumors.—They occur most frequently in the cecum, sigmoid flexure, rectum, and hepatic and splenic flexures. They may cause dislocation of the transverse colon, and the mass be felt a short distance above the symphysis. In most cases the *tumor is not of very firm consistency, is movable, and pits on pressure.* On the other hand, it may be nodular, hard, or angular.

In some cases the bowels may move every day, there evidently being a free central passage.

Gersuny's Adhesion Symptom.—If the abdominal wall over the tumor is gradually depressed with the finger-tips, the pressure gradually diminished, and the fingers slowly withdrawn, one can feel the mucous coat of the intestines loosening itself from the feces forming the tumor (*i. e.*, the wall of the bowel separates from the tumor when palpating pressure is relaxed).

If under intestinal irrigations, etc., the tumor diminishes in size, it is evidently fecal. Anesthesia may rarely be required for examination.

Some of these fecal tumors have developed into large size—over 4 pounds or more—and after the colon is dilated, stercoral ulcers, local or general peritonitis, or intestinal obstruction from kinking, compression, torsion, or internal occlusion may result.

Hemorrhoids are a complication. They are described in a special section.

I have referred to *various nervous symptoms that are dependent on constipation.*

Leube describes intestinal vertigo, which he believes reflex and due to pressure on the hemorrhoidal plexuses of the sympathetic, since the palpating finger in the rectum also produced it.

Senator imputed dizziness and vertigo to absorption of poisonous gases, such as sulphuretted hydrogen; while Nothnagel assumes that nervous symptoms are due to absorption of ptomains, thus causing auto-intoxication.

Auto-intoxication I believe to be the correct explanation. In persons of a neuropathic disposition auto-intoxication from chronic constipation is undoubtedly a contributory factor in the production of nervous disease, especially melancholia.

Fecal Fever.—This is generally due to some complication, such as inflammation, local peritonitis, or stercoral ulcer. With infants and young children fever may be caused by fecal accumulation. I have referred to the cases of dementia paralytica with temperature, with disappearance of the latter after bowel action.

Clark holds that chlorosis is the result of toxins absorbed from coprostasis. Hyperchlorhydria is generally present and this would have an influence on the bowels.

Though diarrhea with marked multiplication of the Bacillus aërogenes capsulatus often occurs in common with severe primary anemia, and in some instances the cases present the blood-picture and clinical characteristics of pernicious anemia, yet Herter shows that advanced infection with the gas bacillus can occur[1] without any diarrhea, in fact, with *obstinate constipation.* Stasis is favorable to putrefaction. The possibility, therefore, of stasis (constipation) favoring in some cases the development of poisons having a hemolytic action on the blood should be considered.

Diagnosis.—The irregularity of the movements and their character, occurring in small balls, pencil shape, or in small fragments, and abdominal palpation disclosing fecal masses are diagnostic. One must remember that daily incomplete evacuations may occur.

Fecal masses are found most frequently in the caput coli, sigmoid, and the rectum. It is always well to examine the latter. *All possible causes of constipation must be considered.* If it is suspected to be due to anomalies of the functions of the stomach, gastric analysis must be performed and treatment directed toward the gastric condition.

Diseases of the heart, lung, liver, and kidneys must be treated if present. Rectal examination may disclose hypertrophy of Houston's valves; in such event, their division by the application of Gant's valve clamps is indicated.

Prolapse of the sigmoid may be determined by inspection with the sigmoidoscope. Gant's suspensory operation is then indicated. Splanchnoptosis if present must be treated and Rose's belt applied.

If none of these causes can be determined, the constipation must be purely functional (habitual), either of the atonic or spastic type.

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[1] Bacterial Infections of the Digestive Tract, p. 207.

With atony of the bowels there are slight bloating, evacuation of hard fecal matter, often in balls covered with a thin layer of mucus, at times dizziness and nervous symptoms. *Severe pains are rare.*

With enterospasm there are uneasiness and pain at the time of evacuation or just preceding it, and at times spells of faintness. The fecal matter is not so hard and is evacuated after considerable straining in narrow tape-like or pencil-shaped pieces. There is no feeling of complete relief after evacuation, but as if more were present. The abdomen may at times be sunken and retracted and the intestinal coils can sometimes be palpated.

Prognosis.—This is favorable in the majority of cases as regards life, though occasionally incurable lesions, such as diverticulitis, ulceration, or even peritonitis and death may result. These complications are comparatively rare. The prognosis as to cure depends on the cause of the constipation. In the functional cases of habitual constipation cures result, but many cases require more or less care for the balance of their lives.

Treatment.—*General Methods.*—The cause of the constipation should be treated. Persons who for years have habitually had a movement every second or third day and are in good health should be let alone.

Prophylaxis.—Never place a patient on a restricted diet for too long a period, excluding vegetables, fruits, starchy foods, and fats which would dispose to constipation. Abuse of cathartics should be avoided.

A hygienic method of living, proper out-of-door life and exercise, diminution of strain and worry are necessary. As few purgatives as possible should be employed.

Training of the Patient.—One should allay the patient's anxiety. He should be told not to worry if the bowels fail to act for a day or two. Meanwhile, rational methods should be undertaken by the physician to produce the desired result. Trousseau first advocated teaching regularity. The patient should be taught to endeavor to have an evacuation at a regular time every day, preferably half an hour after breakfast. He should go to the closet and try to have a passage, but should not exert himself for over five minutes. It is an excellent procedure to aid the return to the habit by insertion of a small gluten or glycerin suppository or, preferably, the injection of 1 to 2 ounces (30.0–60.0) of olive oil with a soft-rubber hand syringe. This is better than waiting twenty-four hours, as the desire is often thus stimulated. Regular habits could thus often be cultivated and the small injection then stopped.

Diet.—The main object is the ingestion of foods which increase intestinal peristalsis and the avoidance of constipating material.

A glass of cold water or, in some cases, of hot water should be taken on rising. Water should be taken on the fasting stomach and a moderate amount—℥viij (250 cc.) at least—of fluid at meals.

Total abstinence from liquid at meals tends to constipate. The following are of value: buttermilk, cider, sour milks, fermented milk, such as lactone-milk, kumyss, matzoon, bacillac, carbonated waters, raw fruits, such as grapes, oranges, grape-fruit, apples, prunes, pears, peaches, plums, mandarins, gooseberries, currents, strawberries, raspberries, blackberries; cooked fruits, honey, lemonade, vegetables rich in cellulose, cabbage, cucumbers, spinach, green peas, cauliflower, green salads, syrup, sugar, salmon, sardines, herring, rye bread, Graham bread, brown bread, pumpernickel; fatty and highly seasoned foods, plenty of butter, cream. Excess of potatoes or rice constipate. Oatmeal is often of value. Milk is constipating with some and purgative with others. Whortleberries are constipating. Red wines, tea, chocolate, and cocoa should be avoided. Beer and champagne are recommended by some. Chicken and red meats are given in moderation.

We may say the diet should be mixed, with a preponderance of vegetable food. Some patients, of course, have idiosyncrasies to certain foods, and one would not give a patient with a delicate stomach cabbage, cider, and brown bread. If the intestines are already overburdened with too much ballast and excess of cellulose has been given, such articles must be restricted.

The internal administration of *olive oil*, ℥j to ij (30.0–60.0) or more, has often an excellent effect; to be given once or twice daily.

If fermentative or putrefactive processes are present, they must receive attention.

In very obese patients one would not give excessively fattening food. Often a few prunes with the morning glass of water and fruit for breakfast are serviceable. The administration of a raw apple thoroughly masticated at 9 P. M., and an hour later a large glass of Vichy, as suggested by Lewis K. Neff, I have often found valuable, a morning movement resulting.

Physical Methods.—These are useful to strengthen the bowel and promote better action or to directly stimulate peristalsis.

Massage.—This is of use in the atonic cases, but not as much so in spastic constipation. It should, preferably, be administered by an expert and the treatment carried out for many months.

Abdominal massage should be carried out in the course of the colon by short tapping motions (vibratory), or by kneading and rubbing. It is preferable to massage from the caput coli to the sigmoid. The small intestine should also be manipulated as well as the abdominal muscles.

Illoway[1] recommends massage for five to fifteen minutes in adults and three to five minutes for children, at least every other day for a period of six weeks, and then, if there is improvement, at longer intervals, but for a long period of time. It should be given preferably early in the morning in the fasting condition.

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[1] Constipation in Adults and Children.

Automassage.—The patient sitting upright with the right hand should stroke the abdomen from the caput coli to the hepatic flexure, and then along the transverse colon. With the left hand he can then massage down the descending colon. Circular stroking movements should then be made over the median abdominal region. I often have the patient follow this out while endeavoring to have the morning defecation. Séances should last about five to ten minutes.

Cannon-ball Massage.—A 3- to 5-pound cannon-ball rolled over the abdomen in the course of the colon and small intestine spirally is of value. The patient can employ this in the dorsal position.

The hollow *wooden* cannon-ball with a screw cap, arranged so that shot can be placed therein, and thus different weights employed with the same ball, is an excellent instrument (Fig. 200). It is arranged with a handle and frame so that it can be more easily manipulated (Fig. 201). One should begin with a weight of 2 pounds and increase it gradually to 5 pounds.

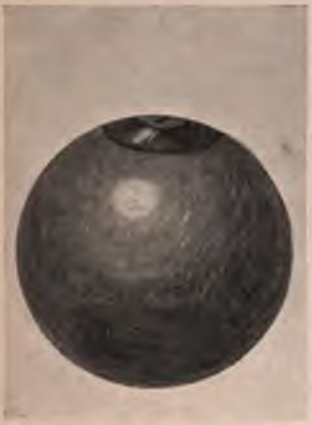
Fig. 200.—Cannon-ball with screw cap.

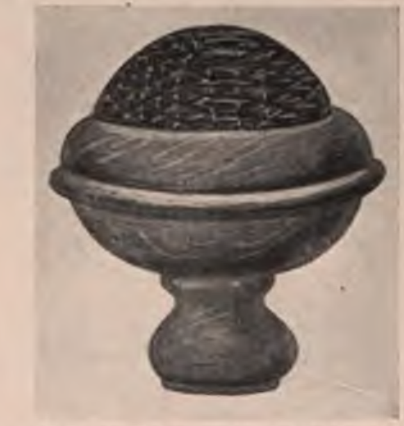
Fig. 201.—Cannon-ball with handle.

Vibratory Massage.—This is of great value. Special vibration should be given over the sigmoid flexure. The Vedee can be used alone or with electricity. Séances should last about ten to fifteen minutes in the course of the colon and over the small intestine.

Hot and Cold Massage Electric Roller.—This instrument has been described and can be employed for the atonic cases.

Gymnastic Exercises.—Exercises which bring the abdominal muscles into play are of value, such as gymnastics on the horizontal bar, horseback riding, hill climbing, skating, rowing, bicycling, golf, tennis, boxing, fencing. Overexertion and too abundant sweating should be avoided. Flexion and extension of the body and lateral rotation while in the sitting posture; bending downward and then upward in the standing position with the knees held stiff; Swedish movements and treatment by the Zander methods are all of value.

Electricity.—Percutaneous electricity, especially faradization over the abdomen, is useful as an adjunct. The intrarectal method (one electrode in the rectum and the other over the abdomen) is recommended, especially galvanization.

The author's method with *recurrent electric irrigation* is practical. With a glass Y attachment and two fountain syringes, alternate hot and cold electric douches can be employed for the atonic condition. I have used both the faradic and galvanic current. In obstinate cases of high impaction I have found hot normal saline douches at 120° F. with the faradic current most efficacious, duration fifteen to thirty minutes.

Kussmaul has suggested one electrode in the stomach and the other in the rectum.

Electricity is indicated in the atonic cases. Static electricity is also recommended. Doumer[1] has employed it in the form of sparks, especially in the left iliac fossa.

Hydrotherapy.—Frictions, cold douches, the alternating cold and warm fan douches, Scotch douches, short cold sitz-baths (five minutes at 12° C.), the wet binder (Neptune's girdle) applied over night, and the Priessnitz compress are all recommended. Hydrotherapy must needs be conducted at a sanatorium.

For practical purposes the sitz-bath and wet abdominal compress suffice the general practitioner.

Injections; Enteroclysis.—Recurrent enteroclysis with hot normal saline solution at 110° to 120° F. for fifteen minutes three times a week, alone or combined with electricity (rectal method) or with the alternating cold douche at 60° F., may prove of service in the very obstinate cases.

Enteroclysis with flaxseed tea has also proved valuable.

The soapsuds and water enema alone—Oj to 1½ quarts (500–1500 cc.)—or with olive oil, ℥viij (250 cc.), or castor oil, ℥j to ij (30.0–60.0), included, or normal salt solution alone, may be required, depending on the conditions.

Often a small enema of normal saline or soapsuds and water of Oj (500 cc.), if given with the patient in the knee-elbow position, is more efficacious than the larger injections. The water injections may be employed at the same hour daily for a considerable time. The large injections, as recommended by some, overdistend the already atonic intestines. The Sims position is excellent for the injection.

Klemperer[2] recommends the injection into the bowel of small quantities of water at bedtime—only ½ pint (250 cc.)—and the patient is told to retain the fluid. It is soon absorbed and evacuation occurs the following morning. These injections are given every night for three weeks, and then every other night.

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[1] Annales d'Electro-Biologie, 1898, p. 722.

[2] Therapie der Gegenwart, 1899, p. 48.

Kussmaul and Fleiner employ an injection of sweet oil into the rectum at bedtime, which is to be retained. I believe it advisable to start with a small quantity, only ℥iv to vj (125.0–200.0), heated to the temperature of the body and slowly injected through a colon-tube from a fountain syringe. The patient should retain the oil as long as possible (over night if he can). Gradually increase the quantity to ℥viij to Oj (250–500 cc.), and in obstinate cases nearly to 1 quart (liter).

As a rule, evacuation follows the next morning. I give the injection every night for a week, then every other night for several weeks, then twice a week, and, finally, once a week. The treatment should cover several months. This method is recommended especially for the *spastic type of constipation*, but I have found it of value in other cases.

Olive oil, ℥j to ij (30.0–60.0), by mouth once to three times a day is a valuable adjunct. Cottonseed oil can be *substituted by enema.*

Glycerin Injections.—Glycerin, ʒj to ij (4.0–8.0), dissolved in ℥iij (95.0) of water and injected into the rectum is of service in some cases, or given as a suppository. It is somewhat irritating.

Flatau[1] inserts or insufflates into the rectum gr. 15 to 45 (1.0–3.0) of boracic acid powder. Bowel action results one-half to three hours later.

Orthopedics.—I have found Rose's belt of value in chronic constipation, even if no ptosis is present. It lends strength to the abdominal muscles and so aids evacuation.

Medication.—In many cases of constipation mild laxatives must be employed, sometimes only temporarily. More powerful cathartics are often required. In constipation of the *spastic form* and in the atonic type with fecal impaction, belladonna is of great value. It should be given preferably as the tincture, in large doses, ♏x to xv (0.592–0.888 cc.), and pushed three or four times a day, so that physiologic symptoms are apparent. In constipation due to atony, strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021) t. i. d., or tincture of nux vomica in 5- to 10-drop doses are of value, even if no laxatives are given. They are often combined with a laxative.

Among the milder laxatives are fluidextract of cascara sagrada and the aromatic fluidextract of cascara, of which the dose is ʒj to ij (4.0–8.0); extract of cascara, gr. 1 to 5 (0.065–0.324); laxophen, a solution of phenolphthalein, ʒj to iv (4.0–16.0); phenolphthalein (purgen),[2] gr. 1 to 5 (0.065–0.324) to gr. 15 (1.0); phenolax (1 to 3 wafers); purgatin, gr. 15 to 30 (1.0–2.0), which is contraindicated in renal disease.

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[1] Berlin. klin. Wochenschr., 1891, p. 231.

[2] Purgen tablets (Bayer), each tablet contains gr. 1½ (0.1) of phenolphthalein; each phenolax wafer contains gr. 1 of the same.

Ad. Schmidt[1] claims that the internal administration of agar-agar, cutting up the straws into small fragments and administering as much as 25.0 gm. a day, aids in softening the feces and also evacuation. He adds to it 25 per cent. of an aqueous extract of cascara sagrada. Schmidt recommends this combination, which is dispensed as "Regulin," as being of value in chronic constipation. Dose, 1 teaspoonful (4.0) to a tablespoonful (16.0) or more, mixed with stewed apples or mashed potatoes. It can be secured as tablets.

Syrup of tamarinds, ʒj (4.0), or a sauce of tamarinds; syrup of figs, ʒj to ij (4.0–8.0); compound licorice powder, ʒj to iv (4.0–16.0). This last gripes some patients. Olive oil by mouth, ℥j to ij (30.0–60.0), several times a day is valuable.

Aloes does not lose its effect even when employed for a long time, and painless defecation results. It may be used alone or in combination. If hemorrhoids are present I do not advise it.

Rhubarb is an excellent drug. Pil. aloës, dose, 1 to 2 pills at night. Tinct. rhei aromatici, ʒss to j (2.0–4.0). Tinct. rhei, ʒj to ij (4.0–8.0). The following are of value:

℞.	Tinct. nucis vomicæ	ʒiss	6		
	Fl. ext. cascara	℥ss	16		
	Pulv. ipecac	gr. iv		26	
	Pulv. rhei	gr. xv	1		
	Sod. bicarb.	ʒiss	6		
	Aq. menth. piperit	q. s. ℥iv	125		—M.

Sig.—Shake. One to two teaspoonfuls (4.0–8.0) t. i. d. in water after meals as a mild laxative.

℞.	Pulv. rhei Magnes. usta Sod. bicarb.	āā ʒiv (16.0).—M.

Sig.—One-half teaspoonful (2.0) t. i. d. after meals.

℞.	Pulv. aloes	gr. xx	1	3	
	Ext. belladonna Ext. nucis vomicæ	āā gr. v		3.	—M.

Ft. pil. No. xx.
Sig.—One to two pills at night.

Podophyllin combinations are quite useful.

℞.	Podophyllin Ext. physostigmatis Ext. nucis vomicæ	āā gr. v	3.	—M.

Ft. pil. No. xxx.
Sig.—One pill at night and in the morning if required.

℞.	Pil. colocynthi comp. Pil. rhei comp.	āā gr. j (0.065)
	Ext. hyoscyam.	gr. ss (0.032).—M.

One pill.
Sig.—One pill before dinner.

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[1] Münchener med. Wochenschr., No. 41, 1903.

℞. Aloin .... gr. $\frac{1}{5}$ (0.013)
Strychnin sulph. .... gr. $\frac{1}{60}$ (0.00108)
Ext. belladonna .... gr. $\frac{1}{8}$ (0.008)
Pulv. ipecac .... gr. $\frac{1}{16}$ (0.004).—M.
One pill.
Sig.—One to two pills at bedtime.

℞. Resinæ podophyllin .... gr. $\frac{1}{6}$ (0.011)
Pil. rhei comp. .... gr. iiss (0.162)
Ext. hyoscyam. .... gr. $\frac{1}{2}$ (0.032).—M.
One pill.
Sig.—One to two pills at night.

℞. Ext. colocynth comp. .... gr. j (0.065)
Ext. jalap .... gr. $\frac{1}{2}$ (0.032)
Resin. podophyllin .... gr. $\frac{1}{4}$ (0.016)
Leptandra .... gr. $\frac{1}{2}$ (0.32)
Ext. hyoscyami }
Ext. taraxaci } .... āā gr. $\frac{1}{4}$ (0.016)
Ol. menth. pepmt. .... q. s.—M.
One pill.
Sig.—One to two pills at bedtime.

Jalap[1] and colocynth belong to the stronger remedies, and I only employ them temporarily to empty the bowels. The same is true of castor oil and calomel.

Hunyádi, Friederickshall, the Homburg Waters, Carlsbad salts, Pluto, Apenta, Rubinat, etc., may be necessary for a brief period, but should not be used for any length of time. In anemic patients with constipation the following pills are of service:

℞. Pill (Blaud iron) .... gr. v (0.324)
Aloin .... gr. $\frac{1}{20}$ (0.032).—M.
One pill.
Sig.—One to two pills t. i. d. after meals.

or

℞. Blaud's iron pill .... gr. x (0.6)
Pulv. capsici .... gr. $\frac{1}{4}$ (0.016)
Aloini }
Strychnin sulph. } .... āā gr. $\frac{1}{30}$ (0.0022).—M.
Acid arseniosi }
One pill.
Sig.—One pill t. i. d. after meals.

Fecal Colic, Fecal Tumor.—It is an error to at once administer large doses of cathartics, and in some cases positive harm may result. The rectum should first be examined and all material found therein removed by the finger and then by enemata. High injections of soapsuds and water, in all, 1500 cc., containing olive oil ℥viij (250 cc.) to 1 pint (500 cc.), should be given, in some cases in the knee-elbow posture, in order to soften and remove accumulation. Frequent injections and irrigations should be given to start movement for the first twenty-four to forty-eight hours. Ox-gall, ʒj to iij (4.0–12.0), with glycerin, ʒij to ℥ss (8.0–16.0), added to the enema are of value.

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[1] Compound jalap powder, gr. 30 (2.0), with calomel, gr. v (0.6), is a good combination.

Olive oil, ℥ij to vj (60.0–200 cc.), can be given by mouth if necessary t. i. d. to soften the dejecta. Later, castor oil, calomel, or compound jalap by mouth, and saline cathartics.

Frequent irrigations, in some cases with electricity, can be added to the treatment. Large doses of tincture of belladonna and strychnin may later be of service. In some cases it takes several weeks for an old accumulation to be completely removed.

Spasm of the Sphincter.—This should always be examined for, especially in cases of spastic constipation. Gradual dilatation or rapid dilatation under an anesthetic are curative. Local disease should be treated.

DIARRHEA

Clinically, diarrhea may be defined as abnormal rapidity of intestinal peristalsis, accompanied by frequent evacuations of the bowel contents, which are too liquid or are watery in character.

Some patients normally pass solid dejecta several times a day, but this is not diarrhea; yet a single solid movement may possess pathologic significance.

Diarrheal stools are caused by the excess of water in the feces, and may be due to the liquid contents of the small intestine being so rapidly hurried into the colon that little absorption is able to occur in the small intestine. Free transudation of water from the blood-vessels or the glands may be a factor.

Rapid peristalsis in both the small and large intestines, or in the latter alone, is another cause.

At times increased peristalsis is the only factor, and there are no chemic or physical changes in the bowel contents and no structural changes in the wall of the gut. Increased peristalsis usually involves the large intestine as well as the small.

Pathologic increase of intestinal peristalsis may be produced in numerous ways. In the majority of cases it is caused by intestinal diseases in which anatomic changes are present, as in intestinal catarrh, ulcers, typhoid, etc. It may be present without any apparent anatomic lesions, as a result of irritants in the contents of the bowel; or when the contents are normal, but the irritability of the nerves of the intestinal wall is increased; or when the muscular coats of the intestines are stimulated by an irritant circulating in the blood or affecting the central nervous system.

Frequently there are several factors. The appearances of the evacuation, both macroscopic and microscopic, in diarrhea vary according to the etiology of the disease and the anatomic changes in the gut, when such are present.

In every diarrhea it is important to know whether it is produced by abnormal transudation or exudation, with increased peristalsis of the large intestine; or whether in addition the *peristalsis of the small intestine is increased.* In the last event large quantities of unchanged digestive fluids and undigested food remnants are evacuated, and nutrition is markedly impaired. Bile-pigment reaction in the feces shows involvement of the small intestine.

We may classify two forms of diarrhea: first, with intestinal lesions; second, diarrhea without lesions. The first group is described in the appropriate sections. The second group, with no intestinal lesions, is classified as follows:

1. Diarrhea due to irritation from the bowel contents. Diarrhea dyspeptica, Diarrhea gastrica, Diarrhea stercoralis, and Diarrhea entozoica are subdivisions.

2. Diarrhea due to irritants transmitted in the blood, such as the uremic type.

3. Diarrhea nervosa (nervous diarrhea), due to irritation of the nervous system.

4. **Diarrhea Cathartica.**—This type belongs in a class[1] by itself. The majority of purgatives stimulate the peristaltic action of the entire intestinal tract. The peristaltic action of the large intestine is chiefly affected, as by the aromatic laxative drugs. The movements in this case are thin and liquid, since increased peristalsis interferes with the absorption of the ingesta and intestinal secretions.

With the alkaline laxative salts the action is not only to increase peristalsis, but they withdraw the water from the blood and stimulate the intestinal secretions. The prolonged use of drastic purgatives or excessively large doses produce an acute catarrh of the intestines.

DIARRHEA DUE TO IRRITATION OF THE BOWEL CONTENTS

Diarrhea Dyspeptica.—Certain articles of diet may produce diarrheal evacuations, such as fresh fruit, cucumbers, pickles, cabbage, turnips, beets, etc. Patients vary as to suceptibility. Milk produces diarrhea in some, while others it constipates. Excess of food or too great ingestion of water or beer with the food may prevent gastric digestion. The ingesta entering the intestines unchanged may cause diarrhea.

Intestinal fermentation or putrefaction, spoiled food, and autointoxication may produce diarrhea. With the last the diarrhea is due to more than the mere local irritation.

In neglected or severe cases of pure dyspeptic diarrhea, long-continued irritation may give rise to true catarrh.

Diarrhea Gastrica.—Einhorn and Oppler first called attention to diarrhea resulting from disturbances of the stomach functions.

Hypochlorhydria and achylia gastrica are the most frequent causes of this type of diarrhea; *more rarely* hyperchlorhydria or motor insufficiency. In hypochlorhydria or achylia, diarrhea with intestinal symptoms such as flatulence, borborygmi, and colicky pains may predominate. The stools are often quite undigested.

These cases, if prolonged, may develop intestinal catarrh.

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[1] This type, thus referred to by Nothnagel, is merely mentioned in passing. Colocynth and aloin in excess may also produce the condition.

Diarrhea Stercoralis.—Diarrhea with constipation. If constipation occurs in a person whose bowels usually are regular, diarrhea may follow the attack of constipation. The diarrhea is accompanied by colicky pains, bloating, and by the development of more or less offensive gases, such as sulphuretted hydrogen, etc.

It is believed that the diarrhea is caused by the development of these gases in the intestinal contents as a result of stagnation of the fecal matter. Hardened fecal matter may irritate the mucosa and produce secretion and peristalsis. With stercoral diarrhea the passages are at first formed, then mushy, and finally liquid. Scybalæ may be found in the dejecta.

The passage of flatus affords temporary relief. Thorough evacuation of the bowels relieves all the symptoms. Neglected cases may cause intestinal catarrh.

Diarrhea Entozoica.—Intestinal parasites, the tapeworm, for example, may in some cases cause persistent diarrhea. Like other types of diarrhea, there are probably at first no changes in the mucosa, but long-continued irritation will produce catarrh.

DIARRHEA DUE TO IRRITANTS TRANSMITTED IN THE BLOOD

Diarrhea due to the hypodermic injection of certain drugs belongs to this class. The diarrheas of septicemia, nephritis, diabetes, cholera, malaria, etc., are explainable by this theory.

DIARRHEA NERVOSA (NERVOUS DIARRHEA)

This type depends on nervous disturbances, without any morbid changes in the walls of the intestines. Trousseau first described nervous diarrhea. No impairment of digestive functions is present.

It originates either from excessive stimulation of the nerves governing peristalsis (the motor function) or from the transudation of serous material into the intestinal canal (secretory function), produced by nervous influences. In some cases probably both factors are concerned.

The stimulus may arise from the nerve centers and be transmitted through the fibers of the vagus, sympathetic, or splanchnic nerves to the intestinal ganglia.

As an example of nervous diarrhea, numerous watery evacuations may occur as the result of some emotion, such as fright or shock, in which cases the stimulus arises in the brain centers. These are more especially acute transitory attacks.

Nothnagel and Peyer[1] report instances of chronic nervous diarrhea: thus, some persons will be attacked with gurgling, abdominal pain, tenesmus, and diarrhea as soon as they find they can secure no access to a water-closet; while with others the sight of the toilet produces diarrhea. Some patients may have attacks at definite hours, without any relation to surrounding conditions.

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[1] Wiener Klinik, 1893, Heft 1.

In others, nervous symptoms precede the diarrhea, such as vertigo, stupor, giddiness, congestion of the head, reddening of the face, hot flushes over the body, fear, oppression, palpitation, rapid breathing, etc. These symptoms often disappear after a few diarrheal movements.

The number of stools varies; these may be from two to four, or even to fifteen, consisting of thin liquid contents, with mucus rarely present. At times the first movement is solid, the next mushy, and the subsequent movements liquid.

Occasionally peristaltic unrest, borborygmi, and severe tenesmus may accompany the movements.

This form of diarrhea is found as a symptom in hysteria or neurasthenia, in the nervous and debilitated, and even in healthy persons after a nervous shock.

With Graves' disease and migraine this type may occasionally occur.

Charcot[1] describes attacks with tabes (intestinal crisis). Peyer speaks of a reflex form of nervous diarrhea found in consequence of abnormal processes in the genito-urinary tract; for example, in uterine catarrh, emissions, spermatorrhea, and sexual excess.

Fischl cites a case of diarrhea which persisted for several years and resisted all treatment. Replacement of a reflexed uterus cured the case.

Vicarious diarrhea in pregnant women of the neuropathic type has been described by Condio. The diarrhea takes the place of vomiting.

Nervous diarrhea has also been attributed to excessive smoking.

DIARRHEA FROM EXPOSURE TO COLD AND WET

This occurs after a sudden or severe chill from exposure to cold, or wetting of the surface of the body, especially the feet or abdomen. Probably it is due to reflex irritation, transmitted from the cutaneous nerves.

Accelerated peristalsis of the intestines occurs, whether due to reflex stimulation or secondary to hyperemia, it is uncertain. This type of diarrhea is usually transitory. At times it may assume the character of true intestinal catarrh.

TREATMENT OF DIARRHEA

The method of treatment depends on the cause. In the cases with anatomic lesions in the intestines, regulation of the diet and medication appropriate to each special type should be carried out. These methods are described under their special sections, such as under Intestinal Catarrh, Dysentery, etc.

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[1] Prager med. Wochenschr., 1891.

With diarrhea due to a laxative, heat to the abdomen and opium are indicated. Pilulæ opii, gr. j (0.065), three or four times a day, or one of the other opium preparations, or

℞. Tinct. opii ....ʒiiss | 10
Bismuth. subnit ....ʒiij | 12
Mist. cretæ ....q. s. ℥iv | 125.—M.
Sig.—Shake. Two teaspoonfuls (8.0) four times a day.

With **dyspeptic** and **stercoral diarrheas** thorough removal of the sources of irritation are indicated, such as the use of calomel or blue mass, gr. v (0.3), castor oil, ℥iss (45.0), or a saline cathartic, such as magnesium sulphate, ℥ss to ℥j (15–30 gm.), or Sprudel salts, apenta, etc. Intestinal irrigation is indicated in these types.

For **intestinal parasites** an appropriate remedy and a cathartic.

For Diarrhea Gastrica.—Treatment should be given for the existing condition in the stomach; for hypochlorhydria, stomachics, dilute hydrochloric acid, intragastric faradization, etc.

For **achylia gastrica,** chiefly vegetable food, finely divided, and the methods employed for this condition.

For hyperchlorhydria, a rare cause, diet and the alkalis, etc., are indicated.

For Diarrhea Due to Irritants Transmitted in the Blood.—The cause should receive treatment, thus, nephritis, malaria, etc. The general condition should be improved.

Heat locally to the abdomen, liquid diet, rest in bed, the bismuth and astringent preparations, such as bismuth subnitrate, gr. 30 (2.0) t. i. d., bismuth salicylate, gr. 10 (0.6) t. i. d., or bismuth subgallate, gr. 10 (0.6) four times a day; or tannalbin, tannigen, tannopin, or tanocol, gr. 10 (0.6) each, three or four times a day.

Opium preparations may be required, but should be used with caution.

Nervous Diarrhea.—If this depends on reflex action, such as from uterine disturbance, etc., the primary affection must be treated.

In other cases the general condition of the patient must be built up. Neurasthenic and hysteric conditions must receive special treatment. Constipating food may be administered.

Iron preparations, such as iron tropon, ʒj to ij (4.0–8.0), t. i. d.; peptomangan (Gude), ʒj to ℥ss (4.0–16.0), in water t. i. d.; or Fowler's solution of arsenic, ♏v (0.296 cc.), or smaller doses t. i. d.; or sodium arsenate, gr. $\frac{1}{50}$ to $\frac{1}{25}$ (0.0013–0.0026), are of value as tonics.

℞. Blaud's iron pill ....gr. v (0.6)
Sodium arsenate ....gr. $\frac{1}{50}$ (0.0013).—M.
One pill. Make 30 such pills soft with honey and silver coat.
Sig.—One t. i. d. after meals.

The bromids of sodium, ammonium, or potassium, or bromid of strontium, given for a few weeks, gr. 15 to 30 (1.0–2.0) t. i. d., lessen irritability.

Bismuth subnitrate or salicylate, in dosage already given, silver nitrate, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), and the astringents noted above are useful. Heat should be applied.

Opium and its derivatives are generally recommended for this, as well as other types of diarrhea, and are preferable to morphin.

The general tendency to at once prescribe opiates in all diarrheas is to be deplored, especially in the nervous type, as the habit is readily gained. If other remedies fail, they may be used with caution. The following (Wm. H. Thomson) is a useful combination for such purposes. The dosage is small:

℞. Pulv. opii }
Silver nitrate } .... āā gr. v (0.3)
Resin of turpentine...................... ʒij (8.0)
Liquor potass............................ ʒj (4.0)
Pulv. licorice.......................... q. s. to make pills soft.—M.
Divide in pil. No. lx.
Sig.—Two or three pills t. i. d.

℞. Tinct. opii camphor }
Bismuth subnit. } .... āā ℥ss (16.0)
Mist. cretæ.......................... q. s. ℥iv (125 cc.).—M.
Shake.
Sig.—Two teaspoonfuls in water every two or three hours.

℞. Tinct. opii .... ʒiij (12.0)
Tinct. catechu comp...................... ℥ss (16.0)
Aq. destil........................ q. s. ad. ℥iv (125 cc.).—M.
Shake.
Sig.—Two teaspoonfuls in water every three hours.

Opium pills or other combinations can be employed.

In nervous diarrhea the intestines should be trained in the normal direction. Suggestion by the physician is of value. The patient should be instructed that after his morning evacuation he should refrain from other movements except when absolutely necessary. Often he can thus control the desire.

CHAPTER XXIII

INTESTINAL CATARRH; ENTERITIS; COLITIS; CATARRHAL SIGMOIDITIS; PROCTITIS; PHLEGMONOUS ENTERITIS

ACUTE AND CHRONIC INTESTINAL CATARRH

(*Synonyms.*—Enteritis; Catarrhus Intestinalis.)

INTESTINAL catarrh is one of the commonest conditions with which we have to deal, and is of importance, since in acute cases in the young or aged it may seriously endanger the life of the patient; while the chronic cases are often obstinate and difficult to cure, and may impair the general health.

It occurs in two types, the acute and chronic, and these in turn may be primary or secondary to some other disease.

ACUTE INTESTINAL CATARRH

(*Synonyms.*—Enteritis Acuta; Catarrhus Intestinalis Acutus; Cholera Nostras; Acute Diarrhea.)

Acute intestinal catarrh is defined as an acute inflammation of the intestines, characterized by pains of considerable severity and accompanied by diarrheal movements containing an admixture of mucus. The disease may attack a portion of the bowel and we may have a duodenitis, jejunitis, ileitis, colitis, sigmoiditis, and proctitis. In many cases the entire intestinal tract is involved. Appendicitis is described in a separate chapter. Though Woodward[1] held that the small intestine may not be involved alone, yet it unquestionably occurs. In many cases acute catarrh of the colon, on the other hand, gives the prominent symptoms, though there is frequently involvement of the ileum. The *inflammation may also be confined to the large intestine.*

Etiology.—*Age.*—It may occur at all ages, and is frequently found in infants and children. Acute intestinal catarrh may be primary (idiopathic) or secondary to some other disease.

Primary acute catarrh is due to the following causes: 1. An excessive quantity of food, so that a considerable portion remains undigested and acts as a source of irritation; heavy and indigestible food; extremely cold drinks, or an idiosyncrasy to certain foods. In others, who are suffering from slight intestinal disturbances, some articles of diet which otherwise would produce no difficulty may lead to the development of catarrh. Unripe fruit, tainted meat, milk or fish, or vegetables that are overripe or spoiled. Auto-intoxication with diarrhea may result from ingestion of such material, and catarrh is frequently produced if the source of irritation is not immediately removed.

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[1] Medical and Surgical History of the War of the Rebellion.

2. Chemic irritants, both organic and inorganic substances, such as colocynth, croton oil, jalap, senna, podophyllin, spices, pepper, copaiba, mustard, garlic, cantharides, mercury, arsenic, lead, copper, tartar emetic, phosphorus, antimony, alcohol, chloroform, ether, and some of the alkaloids, such as colchicin, veratrin, acids, and alkalis. With concentrated irritants the mucosa may be permanently damaged. Catarrh is caused when smaller quantities are ingested.

3. Mechanical irritants, such as hardened scybalæ, enteroliths, biliary calculi, foreign bodies, such as seeds, fruit pits, coins, etc.

4. Exposure to cold or high temperature, especially in children and infants, or sudden variation in temperature, wetting the feet, are predisposing causes; these conditions probably favoring the development of micro-organisms and producing circulatory change.

5. Chemic irritants from the blood, such as in catarrhal nephritis, catarrh from mercurial inunction, or from abdominal burns.[1]

Acute intestinal catarrh may be secondary:

1. To general infection, as in typhoid, dysentery, cholera, sepsis, influenza, pneumonia, scarlatina, measles, malaria, rheumatism, or other infectious diseases.

In dysentery and typhoid the ulcerations are in part responsible.

2. Direct action of micro-organisms, as in infantile catarrhs due to the activity of numerous types, such as proteus vulgaris and streptococci, Bacillus enteritidis sporogenes,[2] colon bacillus, also Bacilli dysenterii.

3. Extension of inflammatory process from adjacent parts, as in peritonitis, invagination, hernia, tubercular or cancerous ulceration, and thrombosis.

4. Diseases of the liver, heart, and lungs due to stasis and engorgement.

5. In cachexia of cancer, profound anemia, diabetes, Addison's and Bright's disease, intestinal catarrh may be a terminal event.

Morbid Anatomy.—The entire gastro-intestinal tract may be involved or only portions of the intestines are affected. These differences depend on the extent of the catarrhal process and upon the cause and intensity of the inflammation. The anatomic changes are not always commensurate with the severity of the symptoms.

The mucous membrane of the intestines is reddened uniformly or in spots, from light red to dark purple in color, especially marked around the follicles and plaques, on the summit of the valvulæ conniventes and of the villi. If the inflammatory process is intense, extravasations of blood occur. The mucous membrane is swollen and edematous, and is often covered with tenacious glassy mucus, stained by bile or blood and more or less opaque. Desquamated epithelial cells and occasionally a few pus-cells are seen in the mucus under the microscope; layers of epithelium may desquamate and form gray shreds. Fecal contents are usually liquid. The villi and solitary follicles are swollen and appear as whitish nodules surrounded by a red injected area (enteritis follicularis seu nodularis).

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[1] Elimination of the poisons through the bile and from the blood during intestinal secretion is the probable cause of the catarrh.

[2] This was believed to be possibly an impure culture of the Bacillus aërogenes capsulatus, though possibly it is a distinct organism (Herter, Bacterial Infections of the Digestive Tract).

If the process continues, these nodules may rupture and give rise to follicular ulcers. Catarrhal ulcers are produced by loss of the epithelial covering and extension of the inflammation. Irritation in the neighborhood of these ulcers may in protracted cases give rise to polypoid growths.

Microscopically, there are congestion and distention of the blood-vessels of the mucosa and submucosa and small extravasations are at times seen between the glands of Lieberkühn. The spaces between the glands are frequently widened and contain abundant masses of round cells. They are also present in the superficial or deeper layers of the submucosa.

The swelling of the solitary follicles is due to proliferation of their cells and to round-cell infiltration. This is also true of Peyer's patches when they are involved, which is rare to any extent. The epithelium of the mucosa is detached, especially in the large intestine, but this is believed to be chiefly a post-mortem change.

The epithelium is undoubtedly involved in the catarrhal process, as degenerated epithelial cells are found in the mucus with the stool.

The cells of the glands may be cloudy and swollen.

Crypts of Lieberkühn.—The glands are enlarged or the fundus is wider than normal, the opening is narrow, so that the crypt becomes bottle shaped. They may be detached from their base and raised, or protrude into the intestines, or even desquamate.

The submucosa is hyperemic. The muscular and serous coats are unaffected.

Symptoms.—They depend on the etiology, the location of the catarrh, and its severity, so that considerable variation occurs. The general symptoms of an ordinary primary attack are as follows:

It usually begins with a feeling of fulness in the lower part of the abdomen, with attacks of colicky pains and diarrhea. Nausea and vomiting may be associated with these symptoms at the incipiency of the attack. In the mild cases there may be no temperature, or it may be moderate, while in the severe types there may be a chill with rise of temperature to 103° to 104° F. In some cases there may be tympanites. There are gurgling sounds (borborygmi) and the abdomen is tender on pressure, at times over special regions markedly so. Loss of appetite occurs.

The number of stools depends upon the severity of the case. There may be only two or three movements in twenty-four hours, or as many as fifteen to twenty evacuations. The first one or two movements usually contain fecal matter and are somewhat mushy in character. They rapidly become semifluid, and finally thin and liquid. Feces and scybalæ may be found later. The more the colon is involved, the greater is the diarrhea. Diarrhea does not always occur if the small intestine alone is the seat of inflammation. The early stools are frequently of a dark brown color sometimes of offensive odor, the latter condition being especially noticeable if dietetic errors are the cause.

The amount of material passed exceeds the quantity of food ingested, due to the digestive secretions and catarrhal secretion, all of which are rapidly evacuated. When the stools consist of watery discharge and mucus there is often little or no odor. They are slightly acid in reaction and foam-like in appearance.

The stools may be light yellow in color, grayish, or even greenish in young children, or colorless, resembling rice-water. These differences in color are dependent upon the location of the catarrh. In the yellow fecal material Gmelin's reaction for bile-pigment can often be obtained, and this is also found in the green movements, demonstrating involvement of the small intestine. The colorless stools most frequently occur in the specific choleraic types.

Mucus is contained in the stools. It may float on top of the dejections in shreds of various sizes and be of glassy appearance, or be stained in various colors, or mixed with the bowel contents and form a jelly-like mass; it may coat the feces or be mixed with it in small amounts; or the movement may consist chiefly of mucus. In some cases the mucus may only be determined by the microscope.

The localization of the seat of the catarrh is shown by the characteristics of the mucus, to be described later.

Microscopically, there are epithelial cells, numerous micro-organisms, mucus, occasionally a little pus and blood, and undigested food particles. Blood is found only in severe cases where there is marked congestion or ulceration, and pus when ulceration is present.

Chemically, peptones and sugar may be present in the dejecta.

Macroscopically, food remnants may be seen with the naked eye for several days, especially if dietetic indiscretion be a factor.

Subjective Symptoms.—In the milder cases, except for the colicky pains, diarrhea, and the feeling of pressure and fulness, the patients may not feel very badly. In more severe cases they may feel chilly, feverish, dizzy, and weak, at times nauseated, and in some cases they may vomit. Tenesmus may be present if the lower part of the colon or rectum are affected. Gas may be expelled. Borborygmi may be audible. With children and elderly persons the symptoms are often pronounced. Collapse may occur. With infants the hydrencephaloid condition may occur, temperature 104° F. or more, sunken fontanels, rapid pulse, cold extremities, collapse, etc.

General Physical Signs.—The abdomen may be bloated, but when gas is expelled the distention lessens or disappears. Splashing sounds can often be elicited. Over the abdomen usually there is tenderness on palpation, especially in the region of the navel, and frequently in the right or left iliac regions, or along the course of the transverse colon; gurgling sounds can often be heard on palpation. In thin subjects peristaltic movements of the small intestine may be visible either before or after palpation.

If there is much gas, there is a tympanitic note on percussion; it may be dull in character if much fluid be present. Large accumulations of gas are not frequent.

Fever.—There may be no temperature or only moderate fever. In severe types the temperature may be quite high (102° to 104° F.). In some cases there are chills associated with the fever, but the temperature has a tendency *to fall after a few days* and does not show *the characteristic steady increase* of typhoid fever. With tainted food or bacterial infection, fever is especially apt to occur. Such cases run an acute course with severe clinical symptoms. Undoubtedly auto-intoxication is a prominent factor in their production.

With gastroduodenitis, *jaundice* is present, and often vomiting.

Urine may become scanty and concentrated, especially in severe cases when there are frequent movements, and there may be found cylindroids, albumin in small amounts, and hyaline casts. Indican is often present, especially if the small intestine is involved.

Rosenbach's reaction (Burgundy red), on boiling urine with nitric acid, is also found. This also shows intestinal putrefaction. Acetone has been found.

Localization of Acute Catarrh.—Involvement of the small intestine alone is more uncommon, and usually associated with gastritis.

I believe in *acute cases* the involvement of the intestines is the more frequent occurrence, though the intensity of inflammation may be greater *in one portion*, especially in the colon. Some believe the large intestine alone is most frequently involved. This is more so in chronic cases. In the rectum local involvement is quite frequent.

Acute catarrh of the caput coli, due to fecal accumulation, at times occurs, and this must be differentiated from appendicitis. The fecal tumor can be generally discovered by the doughy feel on palpation. The acute symptoms subside rapidly under intestinal irrigation and catharsis.

This refers to a simple catarrh and not to a true typhlitis which involves the muscular tissue. Acute catarrh may occur in the sigmoid and should be classified as *catarrhal sigmoiditis*. In sigmoiditis or perisigmoiditis the musculature is also involved. The cases described by Mayor and Leube are evidently of this type. Diverticulitis belongs to this last class. The nomenclature *should be very specific.*

Localized Physical Signs.—An acute duodenitis is usually associated with acute gastritis, and we have jaundice with local tenderness on pressure in the right portion of the epigastric region. Inflammation of the duodenum with local tenderness may occur after cutaneous burns.

Tenderness on pressure (pain), confined to the middle of the abdomen and not laterally, shows the affection to be probably confined to the other portions of the small intestine; but when the small intestine alone is involved, as diarrhea is usually absent, the diagnosis is difficult. The presence of a considerable number of undigested food particles and epithelial cells tinged with yellow bile-pigment in the feces; microscopic mucus mixed with the stool, with rarely a small amount of visible mucus, are a valuable aid to the diagnosis. Indican is usually present in the urine.

Acute Colitis.—With acute colitis[1] the pain and tenderness are most marked along the course of the colon, over the cecum, transverse or descending colon, sigmoid flexure, or over all together. The stools are diarrheal and contain considerable mucus.

Proctitis is characterized by *tenesmus* and colicky pains in the left iliac fossa. There is a constant desire to defecate. The scybalæ or stools are surrounded with mucus, sometimes tinged with blood, and the mucous membrane may prolapse during defecation and is red and tender. Rectal digital examination is accompanied by much pain, and the examining finger shows, at times, traces of blood.

The *most important method of diagnosis* to localize the process is by examination of the feces, noting the character of the mucus. Macroscopic examination is often sufficient.

When pure mucus is passed without any fecal admixture, catarrh of the rectum, sigmoid, or of the descending colon is indicated.

If small masses of fecal matter or solid balls are passed covered with a layer of mucus, the same condition is indicated.

If there is catarrh of the entire large intestine up to the cecum, even if the movements are thin, shreds of mucus are intimately mixed with the fecal matter, and can be recognized by the *naked eye.*

The close admixture of fecal material and mucus distinguish it from catarrh of the lower colon.

In *catarrh of the upper colon alone* or of the small intestine, or small intestine and upper colon alone, usually no mucus can be seen with the *naked eye,* and hyaline *microscopic* lumps of mucus are found intimately mixed with the stools. Small amounts mixed in the feces are at times visible.

Diagnosis.—If a colon-tube be introduced high into the rectum, and lavage be carried out intermittently with warm water through a funnel, by the same method as lavage of the stomach, the recovered fluid will contain visible mucus, and demonstrate that catarrh of the large intestine is present. This method was suggested by Boas.

The presence of yellow *mucous granules* in the movements have been considered diagnostic of inflammation in the small intestine, though Schmidt and Boas believe them to be structureless remains of muscle substance, casein, or egg-albumen, colored with bile-pigment.

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[1] More properly, acute catarrhal colitis, to distinguish it from dysenteric and other types.

Bile-pigment.—If bile-pigment reaction can be obtained in the stool or in some of its constituents, this indicates an inflammation of the small intestine, and the more marked the reaction, the higher up the involvement. It may be found in the mucus, and this may be stained a dark orange, green, or greenish yellow.

Cylindric epithelium, round cells, or rarely fat (droplets) may be stained yellow.

An acid *reaction of the stool also shows involvement of the small intestine.*

Boas has subjected a filtrate of the feces to the digestion test with a small piece of albumin, and when the result is positive, justly concludes that the condition originates in the small intestine.

Duration.—Mild cases may rapidly recover in three to five days, while severe cases often continue for several weeks. The intestine remains susceptible to irritation for a considerable time, and errors in diet may cause a recurrence of the attack. The condition may become chronic. Constipation may follow the acute attack, and this should carefully be treated, lest habitual constipation develop. The acute attack may never be entirely recovered from, but gradually develop into a chronic catarrh.

Prognosis.—These cases frequently recover within a short time, but in children or very old and enfeebled persons the disease may occasionally prove fatal. The prognosis as to cure depends upon the etiology of the disease; thus, if due to chemic irritants, the condition may become chronic.

Treatment.—*Prophylaxis.*—Particular articles of food or drink known to produce attacks of acute intestinal catarrh should always be avoided. Some are affected by ice-cream and ice-cold drinks, and these should be forbidden. Exposure to cold or wet should be avoided. *Rest in bed should be enjoined.*

When tainted food has been ingested or indigestible or an excessive amount of food, even though there is diarrhea, a laxative should be given immediately to thoroughly empty the bowel.

Calomel, gr. 5 to 10 (0.3–0.6), followed by a saline cathartic, preferably within six hours for rapid effect, or castor oil, ℥j to ij (30.0–60.0), should be given to an adult. Castor oil may be administered in coffee, sarsaparilla, ginger ale, or with orange or lemon juice.

For infants and young children calomel, gr. ss to j (0.32–0.65), in divided doses, or ʒj to iss (4.0–6.0) of castor oil.

If an acid chemic irritant has been taken, then an alkali should be given, and vice versâ. Antidotes should be administered in the case of chemic poison. It is preferable also to administer a cathartic, so as to remove the poison from the intestinal canal.

Enteroclysis with normal saline solution at 110° to 115° F. is indicated in all these cases, employing 1 gallon (4 liters) by the recurrent method, so as to thoroughly cleanse the large intestine.

Calomel is useful when *there are flatulence and indicanuria.* I prefer a fairly large initial dose in these cases, and to repeat it within a few days, to the method of daily small doses. There is some danger of salivation from frequent small doses. Colonic irrigation once or twice a day is of great importance.

These methods, combined with salol or beta-naphtol-bismuth (orphol), gr. 5 (0.3), three or four times a day, with the other bismuth preparations, or with urotropin, gr. 5 (0.3), and sodium benzoate, gr. 10 (0.6), are generally sufficient.

Dilute hydrochloric acid in ♏x (0.59) doses t. i. d. is an excellent adjunct, providing there be no nausea or vomiting, and resorcin, gr. 5 (0.3) t. i. d., may be employed for intestinal fermentation. One of the best remedies for diarrhea is bismuth.

Bismuth subnitrate, gr. 20 to 30 to 40 (1.3–2.0–2.6), given four or five times a day is of service. It may be combined with saccharated pepsin as a vehicle in mild cases in smaller doses.

Bismuth subnitrate, saccharated pepsin, equal parts, ⅓ to ½ teaspoonful every two or three to four hours during the twenty-four hours.

Bismuth subcarbonate, gr. 15 to 30 (1.0–2.0), four times a day.

Bismuth subgallate, gr. 5 (0.3), three or four times daily with the bismuth subnitrate is excellent.

Bismuth salicylate, gr. 5 (0.3), four times a day is a good antifermentative; or ichthoform, gr. 5 (0.3) t. i. d., in combination with bismuth subnitrate. Ichthalbin, gr. 5 (0.3) t. i. d., is useful.

Tannalbin, tannigen, or tannopin, gr. 5 to 10 (0.3–0.6), can be used in combination with bismuth.

℞. Bismuth. subnit.......................℥ss (16.0)
Mist. cretæ.......................q. s. ℥iv (125.0).—M.

Sig.—Shake. One to two teaspoonfuls in water every two or three hours. The same prescription with 10 to 15 drops (0.6–1.0) of tincture opii camphor is useful if the diarrhea continues excessive.

The following represent *single doses* of remedies which can be taken every three or four hours in persistent diarrhea:

℞. Tinct. opii.......................♏x (0.59)
Tinct. kino }
Comp. tinct. catechu }.......................āā ♏xx (1.18)
Aqua destil.......................q. s. ʒij (8.0).—M.

℞. Tinct. opii.......................♏x (0.59)
Mist. cretæ.......................ʒj (4.0)
Comp. tinct. catechu.......................q. s. ʒij (8.0).—M.

℞. Bismuth. subnit.......................gr. x (0.6)
Tinct. opii deodor.......................♏x (0.59)
Aq. cinnamomi.......................q. s. ʒj (4.0).—M.

I prefer, however, *to avoid opiates as much as possible in my treatment.*

Cotoin, 0.06 to 0.1 (gr. 1 to 2); tincture coto, ♏xv (0.888), or paracotoin, in double dose as compared to the cotoin, have been suggested for diarrhea.

Codein, gr. ⅛ to ¼ (0.008–0.016), or, rarely, morphin may be required. Patient should be kept in bed.

Dry or moist heat, or Priessnitz compress, hot-water bag, flaxseed, or milk or potato poultice, hot pieplate, hot salt-bag, compress of hot water, pepper poultice, weak mustard and flour poultice should be applied. If symptoms of collapse, warmth, hot drinks. Camphor hypodermics and strychnin are indicated. Hypodermoclysis may be required.

Enteroclysis is useful to rid the bowel of irritating material, and for treatment of the acute catarrh when located in the colon. It may be given by enema[1] or, preferably, with the recurrent tube.

When there is marked fermentation—

Acetozone	1: 2000 to 1: 1000
Alphazone	1: 2000 to 1: 1000
Peroxid of hydrogen	℥j (32.0) to 2 quarts (liters), or
Salicylic acid (1: 1000)	2 quarts (liters)
Boracic acid	ʒj (4.0) to 2 quarts (liters)
Permanganate of potash	1: 3000.

Irrigate with 2 or 3 quarts (liters) with a recurrent tube once a day.

Soothing irrigations are flaxseed tea, gum-arabic solution in water at 110° F., slippery-elm water, or normal saline solution.

Occasionally astringent irrigations may be necessary.

First wash the bowel with warm water, then inject tannic acid, gr. 30 (2.0) to 1 liter (quart), and 15 drops (0.888) of laudanum, and hold a while. Weak nitrate of silver, gr. 5 (0.3) to 1 quart (liter), has also been recommended, but is better in chronic cases. Gastritis, jaundice, and malaria should be treated, if present, or any disease to which the intestinal catarrh is secondary.

Diet.—Cold drinks should be avoided. Sanatogen is useful.

Warm teas, such as chamomile, fennel, anise seed, or plain tea and gruels (barley or rice), with or without milk, very dilute milk with lime-water, etc., should be given. Milk is often indigestible and barley broths preferable.

Later, bouillon, water soup (bread softened in hot water with butter and salt), yelk of egg or white, or entire egg (raw or soft boiled), stale bread, boiled water, etc. Avoid *carbonated waters.*

As soon as the diarrhea is over, soft-boiled eggs, calves' brains, scraped beef, mashed potatoes, cocoa, weak coffee, chicken, chops, steak, stale bread and butter, potatoes mashed or baked can be given. Fruit, green vegetables, hot breads, fat, and acids should be avoided for a considerable period.

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[1] About 1 to 1½ quarts (1000–1500 cc.) should be used by enema; hips elevated, injecting with a long tube.

CHRONIC CATARRH OF THE INTESTINES; CHRONIC COLITIS

(*Synonyms.*—Enteritis Chronica; Chronic Enterocolitis.)

This disease is characterized by a chronic inflammation of the mucous membrane of the intestines, which gives rise to various functional disturbances of the bowels. Any portion may be affected.

Etiology.—Chronic intestinal catarrh may arise from an acute enteritis which shows no tendency to recovery, or more frequently from repeated attacks of acute catarrh which follow each other at short intervals before complete recovery occurs. Patients frequently pay no attention to an apparently mild attack and disregard the rules of diet prescribed. As a result, the condition becomes chronic.

In other cases, however, chronic catarrh may have an insidious onset from the beginning. Fecal accumulation, notably scybalæ, may be a cause of a subacute or chronic catarrh. Pressure of tumors narrowing the canal, as pressure from fibroids of the uterus, or adhesions, may be factors.

The direct causes of chronic enteritis are the same as in the acute type, and chronic catarrh may be either primary (idiopathic) or secondary to other affections. For the complete etiology the reader should refer to the causes of acute intestinal catarrh.

Chronic catarrh may be secondary to diseases of the lungs, especially tuberculosis, and also diseases of the liver, heart, kidneys, and diabetes. Intestinal parasites, tapeworms, round-worms, seat-worms, etc., may be causes by producing irritation. I recently attended a case of splanchnoptosis, in which there was marked prolapse of the sigmoid flexure with a tendency to fecal accumulation therein. The patient never had an acute enteritis, but for five years has had discomfort in this region, and invariably every few days the passage of strings of mucus and scybalæ. There were no symptoms of mucous colic. The case was one of chronic catarrh of the sigmoid. The symptoms disappeared under treatment for visceroptosis. The possibility of chronic localized catarrh from visceroptosis, associated with fecal accumulation, is worthy of consideration. Chronic appendicitis is suggested as a cause by G. R. Lockwood, and the cure of chronic colitis has followed appendectomy.

The rectum should always be examined. Pressure on the rectum from a uterine fibroid I have seen produce partial stenosis with coprostasis above and resulting intestinal catarrh. Ulcer, fissure, or hemorrhoids may cause not only local manifestations, but catarrh higher up. I have seen a case with chief symptoms pointing to the descending colon and sigmoid, in which an ulcer high in the rectum was the cause.

Morbid Anatomy.—The anatomic changes in chronic enteritis are similar to those in acute cases, being characterized by hyperemia, swelling of the mucous membrane, and increased secretion of mucus. The color of the mucosa varies from a dark venous red to a pale grayish-red tint; in some cases it may even be gray or slate colored from extravasation of pigment between the glands and at the tips of the villi. The last cases are those of long duration.

The surface of the mucous membrane is covered with a transparent viscid mucus and the epithelial cells are cloudy, in a condition of fatty degeneration, and partly desquamated. In the majority of cases of chronic catarrh the accumulations of round cells (which are characteristic of the acute type) are not seen, but *there is connective-tissue proliferation* in the chronic form.

Exceptions.—In some of the early cases of chronic enteritis the microscopic picture may be much the same as in the acute process; and in other mild cases the only abnormality determined is the accumulation of pigment between the glands or in the muscularis mucosæ and a slight widening of the interstices.

As a result of chronic enteritis, *hypertrophy or atrophy of the intestinal mucosa may develop.*

Hypertrophy of the Intestinal Mucosa.—The glands of the mucosa are elongated, tortuous, irregular in shape, and may form diverticula. Their orifices may become occluded through connective-tissue proliferation and there will be a retention of secretion and the formation of cysts (enteritis chronica cystica). Connective-tissue proliferation leads to the formation of polypi (enteritis polyposa), which are more rare, and are usually found in the large intestine. In many cases the walls of the intestines may become *thickened throughout*, including the *muscular coat,* to the extent of several *times its normal thickness.*

Woodward reports a few cases of proliferation of the intestinal mucosa with its glands.

Atrophy of the Intestinal Mucosa.—The clinical entity of atrophy of the intestines has been in considerable dispute, and undoubtedly a pseudo-atrophy due to post-mortem change occurs.

Riegel, Ewald, and Einhorn believe there is a true intestinal atrophy. In view of the existence of an atrophy of the gastric mucosa resulting from a chronic gastritis and from its occurrence in pernicious anemia, I am convinced that an *atrophy of the intestinal mucosa* may occur in advanced cases of chronic intestinal catarrh. The condition I believe to be rare. Such atrophy may originate in the glandular tissue in the glands of Lieberkühn. There may be an infiltration of round cells, a fatty degeneration, a disintegration, and desquamation or atrophy. On the other hand, it may result from a connective-tissue proliferation compressing the glands.

Musgrave believes an atrophic condition of the intestines may follow the chronic catarrh occurring with amebic dysentery.

The villi degenerate with the atrophy of the glands, shrink, and become small. No ulceration of the solitary or agminate follicles occurs, and it is a question whether atrophy to any extent ever takes place in them.

There is a *degeneration of the muscular coat* and *some thinning* of it. Jürgens[1] has described a fatty degeneration of Meissner's and Auerbach's plexus and of the muscular tissue, as a special type of intestinal atrophy; Sasaki records 2 similar cases dying with the clinical symptoms of pernicious anemia. These conditions probably are related to intestinal atrophy.

Atrophy of the mucosa occurs more frequently in the colon, especially in the cecum, ascending colon, or ileum near the valve. Large portions of the intestines are rarely found atrophied, but the process generally involves certain parts.

Ulcerative Processes.—As in acute enteritis, we may have *ulcerative processes in chronic catarrh of the intestines.* Superficial erosions of the mucosa may become deeper with the production of ulcers. Rarely they increase sufficiently in depth, and result in erosion of a blood-vessel with hemorrhage, or cause a local peritonitis with or without abscess, or even a perforative peritonitis. Generally the ulcers remain unchanged for a considerable time or cicatrize; occasionally stricture of the intestines may result. The follicles may occasionally swell up and burst, producing small follicular ulcers. Frequently healing takes place. *Extensive ulcerations are seldom met with in chronic enteritis, unless accompanying a tubercular process.*

The "sago grains" or frog spawn in the feces, formerly believed pathognomonic of follicular ulceration, are shown to be of vegetable origin.

Kitagawa finds that some of these grains are mucous in character, but this in itself militates against ulceration, as ulcers do not discharge mucus, but pus. The presence of mucus merely indicates catarrh.

Symptoms.—The chief diagnostic symptom of chronic intestinal catarrh is the *abnormal character of the feces.* It seems advisable to first describe the subjective and objective symptoms which occur in many cases. Some patients complain of no subjective symptoms whatever. The majority of cases complain of a feeling of discomfort or occasionally of slight pains in the abdomen. These symptoms are apt to be most marked after eating, usually several hours; or frequently just before or even after defecation. At times these sensations may disturb the patient an hour or two before rising.

There is a feeling of tension or bloating of the abdomen which may be relieved by the passage of flatus, and this tendency gives rise to considerable annoyance. Flatulence may become so severe as to cause shortness of breath, an asthmatic attack, palpitation, angina pectoris, or cerebral congestion and vertigo, all of which symptoms are alleviated by belching of gas. Flatulence is much more characteristic of chronic enteritis than of the acute type.

Borborygmi are often present. Severe pains are usually absent, though slight colicky pains of rather brief character may be present.

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[1] Berlin. klin. Wochenschr., 1892, p. 357.

These are often relieved by bowel movement or by the expulsion of flatus.

In some cases the general health is not impaired; while in other cases it is undermined. General nutrition may become impaired, especially if the small intestine is also involved. In the latter case gastric symptoms, such as anorexia and nausea, and occasionally vomiting, may be met with.

The patient may feel weak, disinclined to work, be irritable, depressed, and even hypochondriac or melancholic. There may be loss of weight, anemia, slow pulse, cold extremities, and attacks of severe headache. The nervous symptoms are due in part to depression from an evidently chronic and persistent disease, and to a large degree to auto-intoxication.

Physical Signs.—The abdomen may appear distended, especially a couple of hours after eating, with some tenderness on pressure. In chronic enteritis the colon seems to be more usually affected.

Chronic Catarrhal Colitis.—There may be *chronic catarrhal colitis*, with tenderness over the caput coli and ascending colon, with the sensation of a hard mass which yields to the examining finger on pressure (fecal accumulation), or this part may be tympanitic and give the splashing sound from gas and liquid. The same may be true over the descending colon, sigmoid flexure, or transverse colon. There is often tenderness on pressure along the entire colon. Pain is felt directly under the point of pressure, or occasionally at a different point further along the colon, due to the passage of gas, which has been forced along by local pressure.

In thin persons peristaltic movements of the intestines are occasionally observed, especially after palpation. In some cases there are no special objective symptoms.

The diagnostic symptom in chronic intestinal catarrh is the abnormal character of the stool—the *presence of mucus.*

The movements are irregular and the consistence of the stool is variable, *but the mucus is diagnostic.* Diarrhea is not a constant symptom.

There are four types of movements which occur in chronic enteritis:

1. In one set of cases there is marked constipation, and a solid movement occurs only once every two or three days and at times only after a cathartic. The feces are usually hard. It is probably due to a diminished activity of the automatic nervous mechanism of the intestines produced by the catarrh (Nothnagel).

2. In others, constipation and diarrhea alternate; there may be hard movements for several days and these succeeded by five or six thin or mushy movements, mixed with mucus, and accompanied by severe colicky pains. These, in turn, will be followed by constipation, and so on. In some instances the evacuations will be fairly normal for several days and then diarrheal movements will

occur and after this constipation. Constipation is the chief feature in these cases. The reflex irritability of the nervous apparatus, however, is quite good, and decomposition of the stagnating bowel contents eventually causes increased peristalsis with diarrhea.

The periods of constipation or diarrhea, on the other hand, may continue for a long time; thus, constipation for several weeks or months, and then diarrhea for weeks or months. Probably in the latter class there is an acute exacerbation of the catarrh.

3. Rare cases, occur in which there is a daily evacuation of unformed and mushy feces.

4. Cases in which for months the patients pass several diarrheal stools each day. The small intestine is involved as well as the large bowel, as there is a bile-pigment reaction, as a rule, or there are yellow fragments of mucus or epithelial and round cells tinged with bile. The food on account of the catarrhal process is not completely digested in the small intestine, and abnormal products, such as acids, etc., are produced, so that the undigested food and fermenting material give rise to increased peristalsis.

In addition there are some in which the nervous element is a factor combined with the catarrh, and movements occur during the night or early in the morning. F. Delafield[1] describes this type.

The *presence of mucus in the feces is characteristic.* The type of mucus in mucous colic (Enteritis membranacea), which occurs in large amount, and the symptoms render the differential diagnosis comparatively easy. In other cases the presence of mucus demonstrates a true catarrh. In habitual constipation there may be a thin shellac-like covering of mucus over the scybalæ, and this appearance is not found in chronic enteritis. With chronic catarrh with constipation, the quality of the dejecta may be nearly normal, except there is an admixture of mucus. In rare cases the mucus may be absent, or it may be very tough and adhere to the intestinal wall, or the scybalæ may be too small to scrape it off. However, on most occasions mucus will be present, and if there is doubt, washing of the bowel by means of the tube and funnel will eventually bring it away.

Besides the presence of mucus in mucous colic, in some cases of intestinal dyspepsia there is mucus in the stools.

Intestinal Dyspepsia.	*Chronic Enteritis.*
Pure mucus *alone.* Stools gelatinous. Mucus microscopic and seldom visible.	Mucus with epithelial and round cells (diagnostic).
Green stools with acid reaction; bile-pigment gives pronounced reaction.	
No fecal odor.	Alkaline stools. Fecal odor.

The quantity of mucus varies greatly; in most cases only a small or moderate amount.

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[1] Med. Record, May 11, 1905.

The various *combinations of mucus* with the stool and the *localization of the catarrhal process* have been described under Acute Enteritis (page 483), to which I refer my readers.

We may have therefore: (1) Chronic catarrhal enteritis (alone), rare; (2) chronic catarrhal enterocolitis; (3) chronic catarrh of various portions or of the entire colon or of the rectum; thus, chronic catarrh of the caput coli, of the ascending, transverse, or descending colon; or chronic catarrhal sigmoiditis, or proctitis. *The colon is most frequently involved.*

When the movements are watery and thin the fecal matter is a light brownish yellow or grayish yellow, and may contain little biliary matter. Undigested meat or starch particles can often be seen in these cases.

Microscopically.—Though nothing may be discovered macroscopically, we may find with the microscope undigested meat-fibers, starch granules and fat droplets, also mucus and *round and epithelial cells*, at times yellow and shrivelled up. They indicate catarrh of the small intestine. Blood is never present unless due to ulcer or hemorrhoids. Pus is rarely found and only as isolated cells.

Dejecta resembling pus dilated with water (Blennorrhœa intestinalis) shows diphtheritic inflammation when pus is seen in large amount under the microscope. Large masses of epithelial cells in various degrees of degeneration are present in chronic catarrh. They are responsible for the cloudiness in the mucous secretion.

The character of the food and of the stool, and whether there is constipation or diarrhea, determine the consistence and reaction of the feces and the degree of fermentation. As a rule, alkaline reaction is present. The presence of fermentation and putrefaction can be determined by the abdominal tension, flatus, and character of the stool, which may be fetid and present a foamy surface.

Fecal material may be placed in a fermentation tube and kept at blood temperature for several hours, and the degree of fermentation or putrefaction will be shown by the quantity of gas in the tube. The method is described under "Testing the Intestinal Functions."

The presence of putrefaction will be shown by indican in the urine, and the Rosenbach reaction (Burgundy red) on the addition of nitric acid and boiling.

A chronic catarrhal enteritis complicated with ulcers will show marked diarrhea with pus and blood in the stool. If the ulcers occur in the small intestine *alone* and there is no diarrhea, pus and blood often *will not be present;* but there will be more severe pain, more marked tenderness on pressure, and the clinical symptoms will be more severe.

The diagnosis of the atrophic type of chronic enteritis is extremely difficult. Some question its possibility. There will be a previous history of intestinal catarrh of long standing. Later there will be diarrhea, no mucus, with gradual loss of weight, and at times symptoms of pernicious anemia. Tuberculosis must be excluded. This condition is more frequent in the young.

Course.—The course of chronic intestinal catarrh is usually very tedious. It may last for many years. There may be periods of perfect health, but there is a tendency to relapses from any slight indiscretion.

Differential Diagnosis.—The method of localization of the catarrh, as previously stated, is the same as in acute enteritis (page 483).

Irregular bowel action, with the presence of mucus in the stools of the *character described, associated with abdominal symptoms of discomfort, suggest chronic catarrh.* The discharge of mucous colic is characteristic, as are also the symptoms. In the mucus which is occasionally found with intestinal dyspepsia there is an absence of *epithelial and round cells* and the stools are green and acid, as I have already stated.

In habitual constipation there is an *absence of mucus* in the movements. With marked fecal impaction I have frequently seen a small amount of mucus in the feces due to temporary irritation. After removal of the impaction and subsequent care of the bowels there is no further appearance of mucus. Impaction if neglected, or occurring in frequent attacks, may be a cause of local intestinal catarrh and, as heretofore noted, occasionally of stercoral ulcers.

With malignant disease of the intestines, enteritis is often associated, but the cachexia is marked and other symptoms of the neoplasm are present. With intestinal ulcers there are marked pains, local tenderness, and pus and blood in the stool. With enteroptosis we may have a prolapse of the sigmoid, fecal accumulation, and chronic catarrhal sigmoiditis. In every case of chronic intestinal catarrh, enteroptosis should be examined for.

Rectal examination should be made in every case of chronic intestinal catarrh, as the focus occasionally starts from the rectum and progresses up the bowel as, for example, from an ulcer. On account of its importance I shall devote a brief chapter to Proctitis.

A uterine fibroid may block the bowel, acting as a ball-valve, and from fecal accumulation above this point a marked catarrh of the mucous membrane may result.

Chronic appendicitis may result from a chronic intestinal catarrh; while in some cases a chronic appendicitis may act as a focus of irritation for a localized chronic catarrh in the cecum. Removal of the appendix is curative in the last type. In certain diseases of the stomach there may be constipation or diarrhea, but the absence of mucus in the stools and examination of the gastric contents will settle the question.

In chronic enteritis of the small intestine alone there are usually gastric symptoms, constipation, yellow tinged mucus in the stools, generally microscopic and well mixed with the feces, and the biliary salts reaction.

In chronic catarrhal colitis there is *usually more constipation* and more or less mucus of a grayish tinge, either covering or on the surface of the feces or in small strings. Pure mucus may at times be voided at the end of defecation if the lower bowel is affected. Palpation is of value in locating the position of the chronic colitis, as the affected area is apt to be sensitive to pressure.

Chronic diarrhea is prominent in cases in which both the small and large intestines are involved, and the mucus may be yellow in color and considerable undigested food is often present.

Prognosis.—This depends on the severity of the symptoms, the duration of the disease, and the physical condition of the patient.

In the very young, very old, or those weakened by some other disease, such as endocarditis or tuberculosis, severe types of chronic catarrh may be accessory in producing a fatal result.

As a rule, the prognosis as to life is favorable, but in the severer cases of long standing it is not so favorable as to perfect cure.

The cases have a tendency to relapse, especially after indiscretions, though they may continue for a considerable time with comparative comfort. The milder cases of not long standing may recover under appropriate treatment.

Treatment.—The cause of the chronic catarrh should be carefully sought out and corrected. If, for example, endocarditis with resulting circulatory disturbances (which are a predisposing factor) be present, this should receive treatment. The Nauheim bath, even though taken at home by means of the Triton salts, would be of value in such cases. If enteroptosis be present, such as in the case I have described, where there was marked prolapse of the sigmoid, fecal accumulation, and local chronic catarrh, treatment by abdominal support (Rose's belt) and increasing the nutrition by the methods described under Gastroptosis would be indicated, in addition to the general treatment for chronic catarrh. I have seen one obstinate case cured by S. Gant by drawing up and suturing the sigmoid to the abdominal muscles. Angulations of the sigmoid should be corrected. If worms are present, they should be removed.

These remarks will sufficiently illustrate the necessity for investigation of the cause of the catarrh. Hygienic and dietetic measures are of great importance.

The patient should regulate his life carefully, not overwork nor be under too great business strain, eat slowly and at regular hours, and live in the open air as much as possible. In the constipated cases considerable exercise is of value. When the diarrhea is marked the patient should keep quiet during its active stage, and in some cases remain in bed until it has passed. Nervous disturbances should be avoided. The patient should exercise care not to wet the feet or undergo exposure to cold, and should be properly protected in rainy weather. A flannel band about the abdomen is of value. Change of scene and climate are often serviceable.

Diet.—The patient should eat at regular hours, slowly, and masticate thoroughly. It is preferable to give small meals more frequently than three large meals, and sufficient nourishment should be administered so that there is an increase in weight.

Fried foods, hot breads, rich pastries, desserts, and indigestible substances should be avoided. Though some allow the very moderate use of light wines, and occasionally beer or ale, in constipated cases, and the use of claret in diarrhea, in my own experience I find that patients with catarrhal conditions of the gastro-intestinal tract do much better by eliminating alcoholic beverages altogether.

The character of the diet is dependent upon whether diarrhea or constipation is present.

In *diarrheal cases*, if the type be severe, milk and lime-water equal parts, or boiled milk, or milk and barley-water, or rice-water in combination often are efficacious; though some patients have an idiosyncrasy to milk. Rest in bed for a brief period may be necessary.

Kumyss, matzoon, bacillac, or lactone-buttermilk are often useful in such cases, though with some these preparations are objectionable, increasing the diarrhea. Sanatogen in ʒj (4.0) doses, also raw eggs beaten in milk, may be of value.

In milder cases of diarrhea the diet may be more liberal. Carbonated waters, lemonade, fruits, salads, acids, cabbage, cauliflower, rye bread, brown bread, ice-cream, pastries, oatmeal, green vegetables, corn and beans, turnips, carrots, beets, radishes, celery, and lobster should *be avoided*. Mashed and baked potatoes, rice, sago, macaroni, bread well baked and toasted, with a moderate amount of butter, cream soups, bouillon, soft-boiled or scrambled eggs, sweet-breads, calves' brains, chicken, lamb chops, lean fish, cocoa, tea and milk, or matzoon (with some) can be given.

The drink should not be too hot or too cold, and an excess of liquid should be avoided.

In constipated cases the diet must be more liberal. In addition to the food mentioned above, fruits, such as oranges, ripe pears, grapes, green vegetables, such as spinach, peas, lettuce, cauliflower, plenty of butter, cream, and fluids are of service.

Cabbage, cucumbers, brown bread, sausages, lobster, mayonnaise dressing, and bran breads should be avoided in these cases also.

The *urine findings* should modify the diet—if indicanuria, less meat or none for a time; if fermentation, less vegetables; if nephritis, an appropriate diet.

Massage.—In cases *characterized by chronic constipation*, gentle massage over the course of the colon or the use of a light cannon-ball is indicated. No pressure should be exerted on the ball, but it should be rolled along the colon for five minutes morning and night. Light vibratory massage is of value under similar conditions.

Hydrotherapy.—Warm salt baths at 98° to 100° F., or Nauheim baths (artificial) eight to ten minutes every other day for two or three weeks, or pine-needle baths, bran baths, or mud baths may be of some service in some diarrheal cases. Cold baths should be avoided in diarrhea. A Priessnitz wet pack over the abdomen is of value when applied on retiring.

Cold showers and cold sponges are of service in nervous cases. Cold sitz-baths and cold douches over the abdomen are of use in constipated cases, but should be preferably carried out at some sanitarium. I have rarely found the latter methods necessary, but use the artificial Nauheim, pine-needle, or bran baths, also the cold compress to a considerable extent.

Mineral Waters.—A methodic course of drinking certain alkaline or saline mineral waters has proved beneficial in many cases. When taken at the springs, the patient is obliged to follow a rational method of life and diet and is free from worry, and thus receives additional benefit. Carlsbad is especially valuable when there is a tendency to diarrhea, and Vichy is next.

In cases of marked constipation, Marienbad is of service, and also the Hawthorne and Congress Springs at Saratoga. Virginia Hot Springs are also to be recommended. Where neither diarrhea nor constipation are prominent, Kissengen or Homberg; with constipation and anemia, Franzensbad and Elster.

Carlsbad water should be taken in small quantities, a wineglassful twice a day, or even in small amounts of the imported salts, gr. 30 to 60,[1] three to five times a day. It is preferable to begin with small doses. Some do badly with this method. If the diarrhea increases so that the patient begins to lose weight, the treatment should be stopped at once.

Medication.—The method of internal medication depends upon whether constipation or diarrhea is the existing condition. As a preliminary it is always wise to begin treatment with a thorough cleansing of the intestines by a single dose of castor oil, ℥iss (45.0), or a good dose of Carlsbad salts or magnesium sulphate.

If there be marked fecal accumulation, it is better first to employ enteroclysis or enemata, to remove the impaction, and then follow with the single cathartic.

In *constipated cases*, fruits, buttermilk, cold water (glass) on rising, stewed fruits, and a regular hour for attempted stool are all rational.

An enema, olive oil, ℥iv (125.0), increasing to Oj (500 cc.) or more, given slowly by a long tube on retiring, and to be retained, is an excellent procedure. It may be necessary to employ soapsuds enema (never over 1 quart) or normal salt solution. Rhubarb pills, fluidextract of cascara in ʒj (4.0) doses, or the same quantity

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[1] An equivalent of 2.0 to 4.0 of the salts.

105° to 110° F. is excellent if there is much pain, or flaxseed tea at the same temperature is useful. Slippery elm solution and gum-arabic solution are excellent soothing applications.

Listerin, glycothymolin, borolyptol, and boric acid, ʒj to ij (4.0–8.0) to the quart, are of service.

Tannin, gr. 10 to 20 (0.6–1.3) to the quart (liter); zinc sulphocarbolate, gr. 10 to 15 (0.6–1.0); or borax, ʒj (4.0) to the quart, are good astringents; salicylic acid, gr. 15 (1.0) to the quart (liter); irrigation once a day or every other day is of value.

I often employ flaxseed tea or gum-arabic one day, and one of the mild antiseptics or astringents the following day.

In obstinate cases, nitrate of silver solution, gr. 10 to 20 (0.6–1.3) to a quart (liter), given once or twice a week, is a valuable adjunct. If the patient complains of pain, a subsequent injection of normal saline solution is of service.

The bowels should be thoroughly emptied, preferably an enema given an hour or two before local treatment.

Protargol or argyrol (1 : 1000 or 1 : 1500) is of service in place of the silver nitrate.

Surgery.—In cases with evident ulceration, if no benefit results from medical treatment conscientiously applied for a year, I would advocate appendicostomy or cecostomy with subsequent irrigations. In catarrh without ulcers I do not believe operation is indicated.

PROCTITIS

The rectum is very liable to bacterial infection, both from within and without, through the anal opening, so that ulcerations and periproctitis may occur. I will briefly refer to this disease, merely to serve as an index to the practitioner.

Etiology.—As this condition is often a part of chronic enteritis, the etiologic factors may be identical. Local conditions may also produce it; thus, traumatism, as by a syringe-tip; sodomy; impacted feces; worms; foreign bodies introduced through the rectum or lodged there during their passage from above, such as fish-bones, pins, etc., hemorrhoids, polypi; prolapse, intussusception; tumors; pressure from other organs; displacement of the uterus; stone in the bladder; inflammation of adjacent organs, such as uterus, tubes, prostate, or seminal vesicles. Sitting on cold stones or wet seats may be a cause. Idiosyncrasies to certain foods seem to be a factor.

Proctitis is classified as follows:

1. Acute simple catarrhal proctitis.
2. Chronic proctitis: Atrophic form; hypertrophic form.
3. Specific forms: Gonorrheal, dysenteric, diphtheritic, erysipelatous, and syphilitic proctitis.

Pathology.—With simple catarrh there is no pus, except possibly a minute amount. With ulceration there is pus and blood.

The acute and chronic types of intestinal catarrh have already been described. It is preferable to examine pus (if such is present) for gonorrhea, especially if there be a gonorrheal vaginitis, and it may even pass by extension from Bartholin's glands.

Erysipelas may extend from without. Pseudomembranes occur with the diphtheritic type. Amebæ, or the bacilli dysenteriæ, are found with the dysenteric type. We may have the primary chancre of syphilis in the rectum, in which case relaxation of the sphincter will also be noted, or there may be secondary ulceration and catarrh. The test for Wassermann's reaction should be made if syphilis is suspected. Actinomycosis may rarely occur.

Symptoms.—They have been described. They are: Marked straining and tenesmus; passage of mucus with the stool, or mucus alone with pus and blood if ulceration is present; frequent micturition; throbbing heat and weight in the rectum. Constipation at first, later diarrhea; heavy and aching pain in the rectum and down the limbs; often pruritus and prolapse of the rectal mucous membrane or hemorrhoids; temperature; coated tongue; abscesses of the rectum may develop.

The chronic cases present less severe symptoms.

Digital examination is painful on account of spasm, and the rectum will be found to be very sensitive and hot to the feel. If the inflammation extend deeper into the tissues, it will feel hard and rigid.

By speculum examination it will be seen to be a deep red, with hemorrhages and erosions. Often the condition is so painful that it is not advisable to use a speculum at first. The chronic cases run a less severe course. Complications: Periproctitis or ischiorectal abscess may occur.

Treatment.—Recurrent rectal irrigation with hot normal saline solution at 110° F., or in other cases with cold saline solution at 50° to 70° F., with the patient in the Sims posture for fifteen minutes' duration, is of value, once or twice a day, to relieve inflammation. Flaxseed-tea irrigation is also useful, or the other antiseptics described under Chronic Enteritis.

Injection of hydrastis, 1 to 2 per cent., or aqueous fluidextract of krameria (J. P. Tuttle), 5 to 20 per cent., several quarts, are of service in some cases.

Carbolic acid solutions *should never be employed.*

In the acute conditions I do not care to employ nitrate of silver at first, though later 1 : 2000 every day or two is of value.

Argyrol or protargol (1 : 500 or 1 : 1000) is less irritating. Tuttle suggests the use of the following by injection to quiet irritation after local treatment.

℞.	Flaxseed tea	℥j (30.0)
	Opium	gr. ss to j (0.032–0.065)
	Aqueous fl. ext. krameria	♏xxx (1.77).—M.

A suppository of opium and iodoform may be substituted.

If there is marked purulent inflammation, then twice daily irrigate with:

Peroxid of hydrogen 8 to 10 per cent., or 1 : 1000 acetozone or alphazone, or even 1 : 10,000 bichlorid of mercury once in twenty-four hours.

The bowel should be irrigated well with saline solution after each movement and medicated solutions used once or twice a day.

Injections of starch and laudanum should only be used once or twice a day in severe cases to relieve irritability.

After the acute stage has passed, S. Gant recommends spraying the rectum with permanganate of potassium (1 : 3000), or with zinc sulphate, copper sulphate, or nitrate of silver, 1 per cent.

In chronic cases irrigation with nitrate of silver (1 : 2000 to 1 : 4000) is of value, every two or three days.

I have seen an excellent result in a severe chronic case from the injection of the aqueous fluidextract of krameria, suggested by J. P. Tuttle. His formula is as follows:

Macerate 1 pound of bark of krameria in a long percolating tube for twenty-four hours. After this a mixture of glycerin (20 per cent.) and water (80 per cent.) is allowed to percolate through it. The percolate should be constantly stirred and refiltered through the bark a second time.

The filtrate is then evaporated down to 1 pound, thus obtaining an aqueous fluidextract containing gram for gram all the therapeutic properties of the bark. The preparation should be kept in a dark place and not exposed to air.

A 10 to 20 per cent. solution of this can be used for irrigation or a local application of it pure.

The diet and internal medication should be the same as described under Chronic Enteritis. Syphilis, if present, should be treated.

Warm sitz-baths aid in relieving pain.

PHLEGMONOUS (PURULENT) ENTERITIS

This disease, a purulent inflammation of the submucous tissue of the intestines, is rare as a primary process. It is probably due to streptococcic infection, the jejunum being most frequently involved. Peritonitis is present, but the purulent enteritis cannot be diagnosed as the cause, in my opinion, until after operation. Phlegmonous enteritis may be secondary to intestinal ulceration, to intussusception, or strangulated hernia. Maragliano has reported septic infection of the ileum probably by the colon bacillus, with hemorrhage, ulceration, and peritonitis.

CHAPTER XXIV

DYSENTERY

Dysentery is defined as an infectious disease characterized by specific ulcerations of the large intestine. In typic acute cases it gives rise to bloody mucous or mucopurulent dejections, accompanied by tenesmus and general symptoms.

Dysentery was known to the ancient world, being first described accurately by Hippocrates 430 B. C., and later by Celsus, Aretaeus, and Galen.

In 1506 the first records of post-mortem examinations of dysenteric subjects were published in the posthumous work of Antonio Benevieni. Following these came many writers, among whom I shall mention a few of the most prominent of recent years: Cruveilhier, Virchow, Woodward, Lambl, Loesch, Kartulis, Quincke, Roos, Musser, Osler, Stengel, Stockton, Harris, Councilman, Lafleur, Ogata, Shiga, Russell, Flexner, His, Barker, Duval, Bassett, Vedder, Musgrave, Strong, Craig, and Thomas.

It has been demonstrated that the disease is due to infections of a specific type—either to the ameba dysenteriæ or to the Bacillus dysenteriæ (Shiga) or one of its strains. The disease is transmitted in the same way as is typhoid fever. It sometimes assumes a diphtheritic type, in which case other bacteria are undoubtedly associated.

The so-called acute catarrhal dysentery, the sporadic form, I believe, is undoubtedly due to the Bacillus dysenteriæ or one of its strains. Diphtheritic dysentery (or, more strictly speaking, *pseudo-diphtheritic, as Klebs-Löffler bacillus is not present*) has been shown in many cases to be due to the Bacillus dysenteriæ. *Undoubtedly mixed infections with other bacteria are found in this type.*

DIPHTHERITIC DYSENTERY

This may be found in combination with amebic dysentery, and, in addition, in some of the amebic liver abscesses numerous other bacteria are present, which demonstrate mixed infection from other sources.

Secondary diphtheritic dysentery is a common terminal event in many acute and chronic diseases; and Vedder and Duval have demonstrated that the Bacillus dysenteriæ is present in these cases.

Diphtheritic dysentery in which the diplococcus pneumoniæ has been isolated has been several times reported. In addition, mercurial poisoning or uremia may have this lesion associated. It is evident, therefore, that other bacteria either present in or entering the intestinal tract under favorable conditions may produce this lesion.

In the ileocolitis of infants dysenteric bacilli of various strains have been discovered, and some of these cases, both clinically and pathologically, present the appearance of an acute catarrh.

Many causes were formerly given for the production of dysentery, but we may say that they only predispose to infection on account of weakening the organism, producing intestinal disturbances. In the case of overcrowding in asylums and camps there is a tendency to unsanitary conditions. By inattention to the proper relation of the latrines, to the water-supply, for example, there may result a severe epidemic.

LOCATION

Dysentery is found in all parts of the world, but is endemic and often epidemic. It is most common in warm climates, such as the southern United States, Cuba, the Philippines, southern coast-line of Asia, Africa, Egypt, Mexico, Central and South America. It has been met with in cold climes, as in North Russia and Greenland; is sporadic in all parts of the United States and occasionally epidemic. Severe epidemics have occurred in the New England States.

During the Civil War, Woodward collected 259,071 cases of acute dysentery and 28,451 of chronic dysentery in the Federal service.

During the recent Spanish-American War the mortality from dysentery and typhoid was far in excess of that from battle; and in the African War the English troops suffered severely. In the Russo-Japanese War the deaths in the Japanese army from dysentery and typhoid were infinitesimal in number, demonstrating that by intelligent care of the water-supply and proper sanitation epidemics of dysentery can be absolutely stamped out.

Dampness, overcrowding, and imperfect ventilation vitiate the system, and so predispose to subsequent infection. With overcrowding and necessarily insufficient and improper attention to sanitary conditions, infection from the dejecta can readily occur, if a sporadic case develop.

Heat and moisture predispose to intestinal disturbances, and readily cause changes in fresh fruits or canned material, if improperly cared for, which in turn produce diarrheal disturbances, and cause susceptibility to infection. Sudden alternations from heat to cold produce the same result. Errors in diet are predisposing causes.

No race or age is exempt from dysentery, and a person going from his native to a warm climate—with the sudden change in food and mode of life incident thereto—is probably more susceptible.

Dysentery is more fatal among the poor and ill-nourished than among the rich, though the latter are not exempt. It is probably more prevalent in epidemic and endemic form in smaller country towns, villages, and farms, where one so frequently sees the well in close proximity to the privy, a stagnant pool, or the family cow-yard.

Dysentery may be endemic, the so-called tropical dysentery; epidemic or diphtheritic; and sporadic (the acute catarrhal dysentery).

It is classified as follows:

1. *Amebic dysentery*, in which there is at times a mixed infection (diphtheritic process).

2. *Bacillary dysentery* (Shiga), or one of its strains, under which are included the sporadic type (acute catarrhal), which probably belongs to this classification, as a catarrhal type (ileocolitis) exists in infants.

The *diphtheritic type* is included and also the *secondary diphtheritic type*, which may be a terminal event in acute and chronic disease, and in which Vedder and Duval have demonstrated the presence of the Bacillus dysenteriæ.

Undoubtedly other varieties of bacteria play a part in the diphtheritic type.

AMEBIC DYSENTERY

(*Synonym.*—Intestinal Amebiosis.)

Definition.—A colitis, latent, subacute, acute, or chronic, caused by the ameba dysenteriæ. There is a special liability to formation of abscess of the liver.

Often these cases occur without the clinical symptoms of dysentery at all. I agree with Musgrave that the condition should be more correctly given the name of "intestinal amebiosis."

The disease is widely prevalent in Egypt, India, the Philippines, West Indies, Southern States, and in tropical countries. It occurs frequently in the United States, and much more in many of our cities than is generally supposed. I have recently attended a case clearly infected in New York State, the patient having never been south. It is endemic, especially in warm climates, and often becomes epidemic. Sporadic cases occur in temperate climates.

In Manila, Strong states that out of 1328 cases in the United States army, 561 were of the amebic type.

At the Johns Hopkins Hospital, Osler reports most of the acute and chronic cases of dysentery were of the amebic variety; during the first fourteen years there were 119 cases admitted, 95 of which came from Maryland—108 males and 11 females.

Source of Infection.—Chiefly from contaminated water or green vegetables and fruit. Musgrave has found the ameba in ice-cream and water-ices.

Amebæ Dysenteriæ.—Lambl first described amebæ in the stools in 1859, and in 1875 Loësch investigated the stools of a dysenteric patient and described the amebæ. He injected the stools into the intestines of dogs and produced ulceration.

Osler, Councilman, Lafleur, Dock, Quincke, Roos, Musgrave, Strong, and many others have carried on investigations.

To obtain a specimen for examination little flakes of mucus or pus should be selected, or the mucus may be secured by passing a soft catheter, or through a speculum. Preferably a saline cathartic should be administered, as suggested by Musgrave, and the fluid portion of the stool examined *while warm.* Swollen, altered epithelial cells must be distinguished from the amebæ. The cells are round with granular protoplasm.

Ameba dysenteriæ is from fifteen to twenty microns in diameter and consists of a clear outer zone, or ectosarc, and a granular inner zone (endosarc), and contains a nucleus and one or two vacuoles (Fig. 202). The movements are similar to an ordinary ameba, consisting of a slight protrusion of the protoplasm. They vary somewhat and can be intensified by having the slide heated. They have a pale green appearance under the microscope. Red blood-corpuscles are at times contained in the amebæ, and occasionally bacteria.

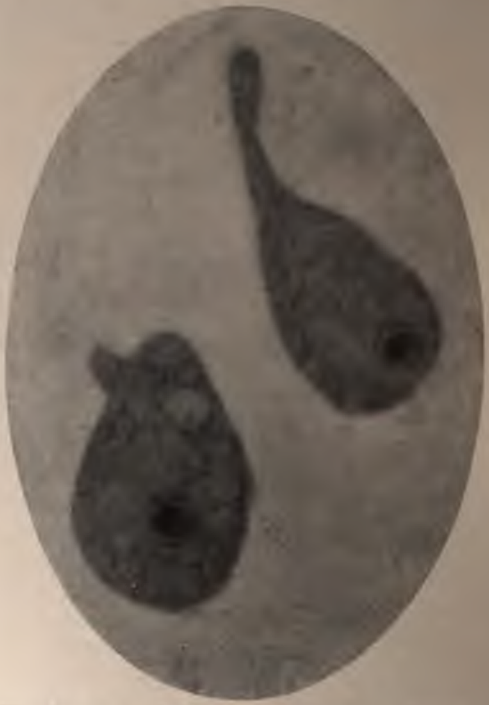

Fig. 202.—Amebæ from a culture. Impression preparation. Borrel's stain (Woolley and Musgrave).

Musgrave recommends Borrel's stain for study of amebæ in the tissues.

They may be in large numbers in the tissues. In the pus of a liver abscess, amebæ may be abundant. In the sputum from a

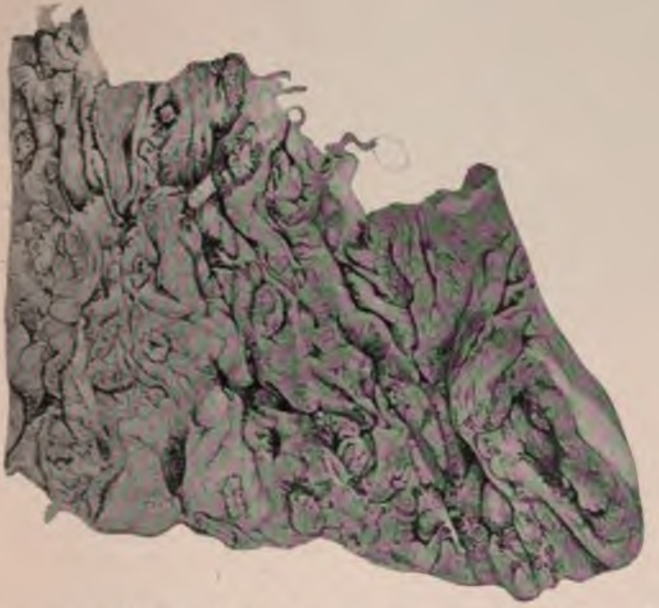

Fig. 203.—Intestinal amebiasis. Cecum. Shows all stages of ulceration. The smallest black points indicate the positions of preulcerative lesions (Woolley and Musgrave).

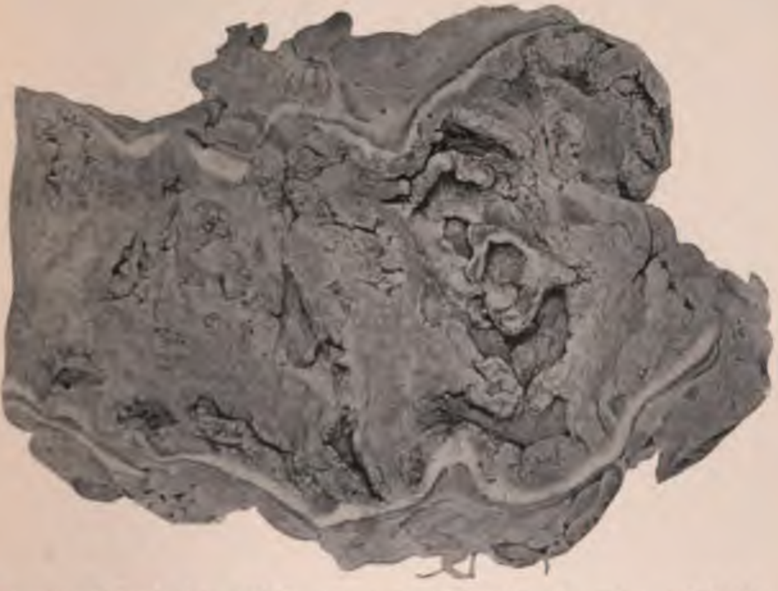

Fig. 208.—Intestinal amebiasis. Rectum. Extensive ulceration and diphtheritis. Thick-walled gut (Woolley and Musgrave).

pulmonary infection from an hepatic abscess they can be recognized.

Quincke and Roos describe three varieties of amebæ in the stools of healthy persons; and Strong two types, only one of which is pathogenic.

Musgrave and Clegg do not think it has been proved there are amebæ non-pathogenic to man. They hold that all such are, or may become, pathogenic.

Amebæ dysenteriæ can be grown in cultures from stools or intestinal ulcers, but not alone as a pure culture. A symbiotic organism is necessary for its development. It has been isolated as a pure culture, in combination with a pure culture of another organism. Fivea, Celli, and Miller claim to have grown it pure, and that it multiplies by division.

Resistant forms of the ameba have been described by Cunningham and Quincke. They are apparently analogous to the gamete forms of the malarial parasite.

The "encysted amebæ" seem, under certain conditions, to be necessary for the transmission of the disease from one person to another, and are regarded by Musgrave and Clegg as the most dangerous type.

Cultures of amebæ have withstood drying for fifteen months.

Location of Lesions.—The lesions are found in the large intestine, rarely in the lower end of the ileum; and abscess of the liver is a common accompaniment, being present in 22 per cent. of Osler's cases.

Pathology of Amebic Dysentery.—*Intestines.*—Though writers refer to the classic undermined type of ulcer in amebic dysentery, three types of lesions are described, which may shade gradually into each other:

1. *Pre-ulceration.*—This stage is characterized by the presence of the "small red dots" of Rogers, varying from 0.2 to 0.5 mm. in diameter, and which are intensely congested (Fig. 203). They consist of capillary hemorrhages into the intraglandular tissue. Erosion of the superficial layers of mucous membrane is usually associated.

There is moderate injection of the mucous membrane and but little thickening of the submucosa. These lesions may be seen in any part of the affected gut, and chiefly in the acute cases.

2. *Ulceration (type of Harris)*, rarer than the classic type, and believed to be intermediate between the petechiæ and "*undermined*" ulcer. They are probably the result of the superficial erosions, and are primarily confined to the mucous membrane, though they reach into the submucosa or to the circular muscle, but no deeper (Fig. 204). They spread laterally. The ulcer has a punched-out appearance and is round or oval, the edges thick and congested. Base is clean, gray, and edematous. They often lie at the apex of

the intestinal fold and tend to increase in the direction of the short axis of the bowel, and are found in all regions, but less often in the advanced or chronic cases. They are most common in the ileum.

3. *Classic or Undermined Ulcers.*—In the early stage they appear as minute yellow or gray spots in the mucosa, at times at the centers of the "Rogers' red dots," and are usually surrounded by a congested area. These spots are the mouths of passages leading to cavities in the submucosa; the mouths and cavities are filled with necrotic material.

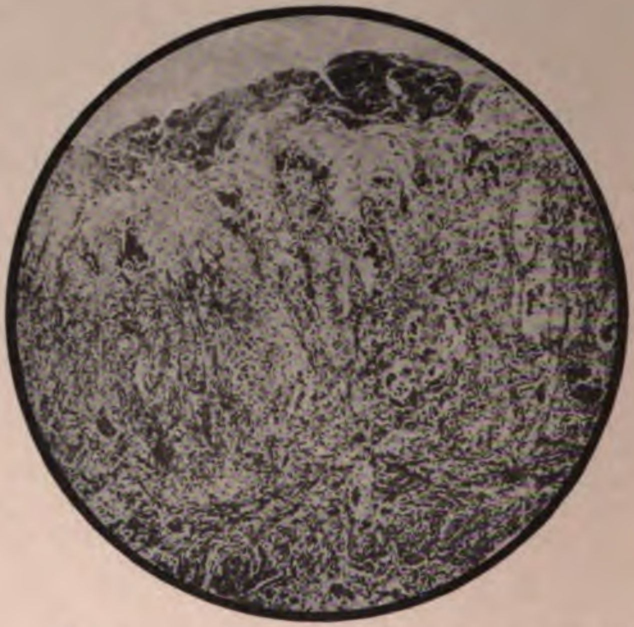

Fig. 204.—Early intestinal lesion. Shows superficial necrosis, glandular distortion, and round-cell infiltration. Borrel's stain. Zeiss obj. A, A, oc. comp. 4; bellows at 30 cm. (Woolley and Musgrave).

As the ulcerative process extends, the cavity in the submucosa is enlarged, and though the necrobiosis eventually involves all the coats, the muscular layers and mucosa are affected less rapidly, and the *latter may be markedly undermined.* The ulcers may gradually coalesce on the surface, or quite frequently the cavities in the submucosa may communicate with each other by tunnels, while the mucous membrane shows a catarrhal condition. The submucosa becomes thickened and edematous, as do often the muscular layers and peritoneal coat (Fig. 205).

The ulcers may be from the size of a pin-head to the palm of the hand. In extensive ulceration the muscular layer may become necrosed or even perforated, and the base of the ulcer be formed by peritoneum or omentum. The omentum plays an *important protective part*, being frequently, early in the ulcerative stage, found adherent to the surface of the intestines. Localized suppuration is, therefore, common. Ulcers may perforate into the subperitoneal or retroperitoneal tissue. They are usually circumscribed, though they may burrow (Fig. 206).

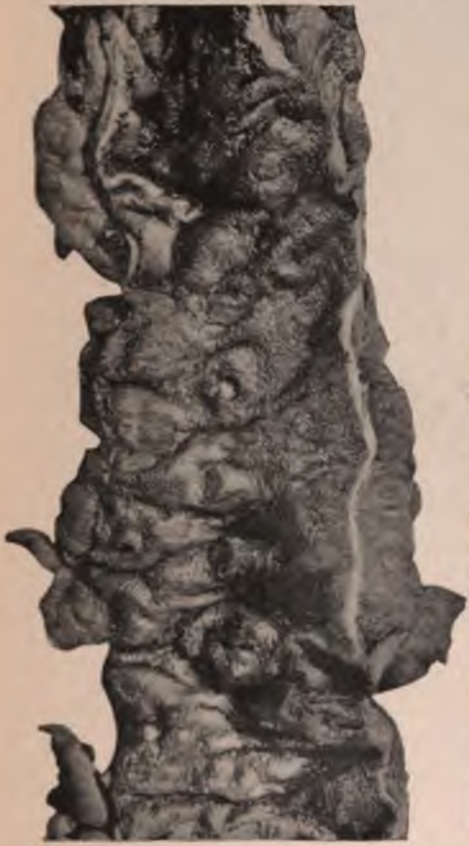

Fig. 205.—Colon. A moderately thickened gut with various types of ulcers (Woolley and Musgrave).

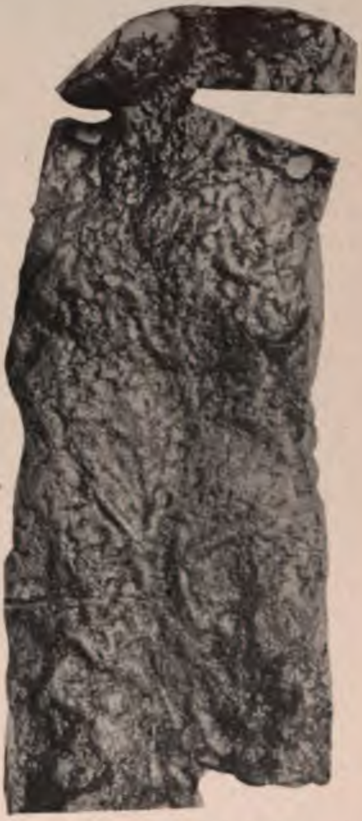

Fig. 206.—Colon. Thin-walled gut, with shallow ulcers, some slightly undermined, others punched out (Woolley and Musgrave).

Healing Process.—In the case of the small ulcers there may be complete repair, the epithelium from the mucous membrane lining the ulcer, except its base. In larger ulcers there may be considerable scar tissue, which may lead to contraction.

According to Musgrave's observation there is established in cases of long duration not systematically treated a chronic *catarrhal condition* with subsequent atrophy (enteritis chronica atrophicans), a thinness of the bowel, absence of normal folds, atrophy of the mucosa and increased length; and he places it in the classification of sprue or psilosis, believing untreated amebiosis to be one of the causes of this condition. There may be, on the other hand, in some cases localized hypertrophy with well-developed polypi.

Location of the Lesions.—Harris believes that in fully one-half the cases the lesions do not extend beyond the beginning of the transverse colon; while Rogers holds that they are more frequently limited to the cecum and ascending colon. The ileum just above the ileocecal valve is *rarely involved*, and generally by only a few ulcers (superficial) and a slight involvement if diphtheritic inflammation complicates. The lower rectum is rarely involved.

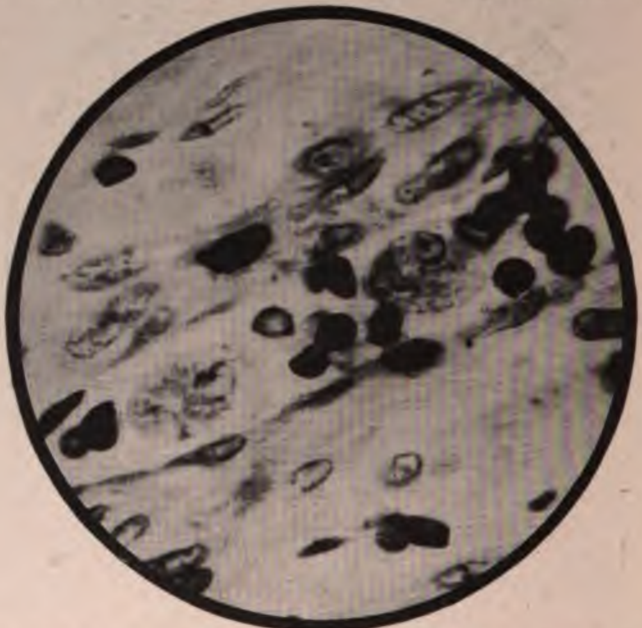

Fig. 207.—Amebæ in a blood-vessel; ×500. Heidenhain iron hematoxylin (Woolley and Musgrave).

Strong and Musgrave, comparing 200 cases treated and untreated, find 159 cases involve the entire large bowel, excepting the lower rectum; 23, the cecum and ascending colon; 2, the transverse colon; the descending colon, sigmoid, and rectum, 9; unrecorded, 7; appendix ulcerated, 2 (amebic); with large intestine, 14; ileum with large intestine, 7.

The presence or absence of irrigation treatment makes a difference. In cases dying early from intermittent disease, ulceration in the cecum and ascending colon, 11; in descending colon, sigmoid, and rectum, 8; in the entire bowel, 6.

Microscopic.—Mucous membrane between the ulcers is little changed. In the neighborhood of the lesions a tendency to *hypertrophy with mucoid degeneration and cyst formation.* In the early lesions, congestion with capillary hemorrhages and edema of the mucosa; increase of lymphoid cells in the interglandular tissue.

Ameba from 4 to 35 μ long are present in the glands, interglandular tissues and blood-vessels, muscularis mucosa, and in the veins of the submucosa (Fig. 207).

In the more advanced condition there is necrobiosis (coagulation necrosis) in the ulcers, with lymphoid infiltration, congestion and thrombosis, and very little polymorpholeukocytic invasion. The amebæ secretion and thrombosis both tend to produce the necrobiosis.

If there is marked polymorphonuclear infiltration, pathogenic bacteria are playing an active part in the process; noticeably then there is diphtheritic inflammation (Fig. 208) or gangrene. When amebæ are found in exudates rich in bacteria, they show evidences of active phagocytosis. In some cases there are many bacteria, but they are probably non-pathogenic, as marked leukocytosis was not present and no process could be seen to be attributed to them.

"In effect, intestinal amebiosis may be said to be rather a subacute chronic inflammatory process, as was demonstrated by the character of the exudate and infiltration, by the early formation of granulation tissue and by the absence of leukocytic infiltration, a notable absence of purulent inflammation. This applies to the cases not complicated by diphtheritic process or gangrene" (Musgrave).

If the diphtheritic process is associated, some strain of the bacillary type is probably responsible; and if gangrene, some *other bacteria* (mixed infection).

Lesions (Abscesses) of the Liver.—There may be local necroses of the parenchyma scattered throughout this organ due to probable chemic products of the amebæ, and also abscesses.

This is quite a common complication of amebic dysentery, and occurred in 27 out of Osler's 119 cases.

Most of the so-called amebic liver abscesses are really local necroses and no pus at all. They may be single or multiple; when single, the right lobe is most commonly affected on the convex surface near the attachment to the diaphragm, or on the concave surface near the bowel.

Multiple abscesses (small and superficial) miliary, containing amebæ, may be scattered throughout the liver substance (Osler).

The hepatic abscess, though it often occurs within the first two to three months after the onset of the dysentery, in some cases may not appear for several years. Some cases are reported as occurring when no dysenteric symptoms had apparently been complained of by the patient.

In the early stage the abscesses are a grayish-yellow color, sharply defined in shape, and contain a spongy necrotic material, with more or less glairy semitransparent fluid in the interstices. The larger abscesses have ragged necrotic walls and contain a viscid greenish-yellow or reddish-yellow purulent appearing material, mixed with blood and shreds of liver tissue.

Old abscesses of a chronic type have a dense fibrous wall. The outer zone is hyperemic; the midzone shows proliferations of connective-tissue cells, compression and atrophy of the liver cells, and an inner necrotic zone. There is the same absence of true purulent inflammation as in the intestines, except where there is a secondary infection with pyogenic organisms. When the latter are present we have true purulent accumulations.

The contents of the necrotic type of abscess show fatty and glandular detritus, necrotic hepatic cells, amebæ, and occasionally Charcot-Leyden crystals. Amebæ are also found in the abscess walls. Cultures are frequently sterile.

Micrococci and bacilli may be found, notably the Staphylococcus aureus, Streptococcus pyogenes, Bacillus coli (with other organisms), Bacillus pyocyaneus, and Micrococcus lanceolatus.

The abscess most frequently points upward and ruptures into the right lung. In some cases an empyema may be produced or a pyopneumothorax. Perforation may occur in other directions, into the pericardium, peritoneum, stomach, intestines, portal or hepatic veins, inferior vena cava, kidney, or externally.

Symptoms.—There has been a tendency to arbitrarily divide amebic dysentery into two clinical types, the acute and the chronic, and to create the impression that in acute and chronic dysentery we have necessarily diarrhea or diarrhea alternating with constipation, the passage of blood and mucus, and the presence of tenesmus. Unquestionably latent and masked infections, with intestinal amebiosis are *by no means rare*, and marked pathologic changes may be present *without objective clinical symptoms*.

Councilman, Lafleur, Osler, Dock, Strong, and Musgrave have reported such cases. The latter notably refers to one case treated six months for constipation, in whom autopsy showed perforation of a liver abscess as the cause of death, with associated lesions (amebic ulceration) of the cecum and ascending colon.

The early recognition of these irregular types is of great importance, especially when amebic dysentery is endemic. Unquestionably, some of our old cases of apparently simple catarrhal colitis are of this type. In fact, I have had one experience with such a case, where amebæ were found present, with ultimate recovery under appropriate treatment.

Since many of the cases may present no clinical symptoms of amebic dysentery, I believe Musgrave's[1] classification, under "*In-*

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[1] Journal of American Medical Association, September 16, 1905.

testinal Amebiasis," to be the most scientific, which I have but slightly modified.

1. Latent and masked infections with the amebæ.
2. Mild and moderately severe infections (subacute dysentery).
3. Severe infection, including gangrenous and diphtheritic types (acute dysentery).
4. Chronic dysentery.
5. Infection in children and in the aged.

Latent Infection.—In these cases there is a pathologic amebic process in the intestines, without any diarrhea or any other symptoms that would indicate the infection.

Musgrave has demonstrated that the symptoms pointing to dysentery may be absent for a long time, yet there are certain symptoms which suggest the condition.

Dull aching abdominal pains are present, which are attributed to catching cold. They first appear and are most active during the night or early morning. Indigestion, headache, lassitude, coated tongue, and a foul breath are present. There are loss of appetite, some loss of weight, loss of color, and at times a yellow skin.

Physical examination discloses on deep palpation tenderness along the colon, especially *over the cecum and ascending colon.* This last is *a significant symptom;* occasionally thickened intestines can be made out. If a hydragogue cathartic be administered, there will be present in the stool amebæ, mucus, tissue elements, and often old blood.

Musgrave has demonstrated by autopsy on his fatal latent cases that the lesions are in the cecum and ascending colon. Any patient, therefore, living where amebic dysentery is endemic, suffering from the symptoms referred to, should be given a saline, and the stool carefully searched for amebæ. The possibility of amebic infection in chronic colitis with no diarrhea should be considered in New York. I have already referred to a patient infected in that State. On the other hand, some of the latent cases, if untreated, may later develop the symptoms of amebic dysentery.

Under the same class of cases (latent) in regions where amebiosis is endemic we may have patients in whom other symptoms, such as chronic constipation, gastric symptoms, or even appendicitis, may mask the intestinal amebiosis. Musgrave has reported such cases.

Mild and Moderately Severe Cases (Subacute Dysentery).—Mild Cases.—These frequently develop from the latent type and often present the aspects of a *diarrhea* and not of dysentery. Abdominal pain, tenderness along the colon, headache, digestive disturbances, irritability, melancholic condition, anemia, and loss of weight are present.

Amebæ are found in the diarrheal movements. Some of these cases never show dysenteric movements, even without treatment. This type Musgrave believes often becomes chronic; while, on the other hand, chronic gastro-enteritis or "sprue" may be the ultimate outcome, giving their clinical pictures. The patient usually dies from intermittent disease or complications.

Moderately Severe Cases (*Subacute Dysentery*).—In these cases with the symptoms just described there may be a diarrhea more marked in the morning, consisting of several semifluid stools, no mucus or blood, and passed without pain. This may intermit with constipation. Finally, the attack may increase in intensity, and mucus and blood will appear. In other cases they will occur from the onset. Usually the more acute the onset, the more rapidly the severe symptoms develop.

Severe Cases (*Acute Dysentery*).—These are the classical cases usually described. Diphtheritic and gangrenous processes, due to some secondary infection, are most common in this type.

The onset is usually quite sudden, whether primary or following on a milder type. There are marked abdominal colic; diarrhea; tenesmus, which may be constant and very painful; straining, and then finally passages chiefly of small quantities of mucus and blood. In some cases the dejecta are hemorrhagic, consisting of pure blood or dark and coagulated blood. Sloughs are passed in others, consisting of gray or blackish masses of necrotic tissue of very foul odor.

The temperature, as a rule, is not high. The patient rapidly emaciates, and the heart becomes rapid and feeble.

Death may occur in severe cases within a week from the onset. Hemorrhage (intestinal) or perforative peritonitis may take place. Many cases recover, but some become chronic. In others extensive ulceration may remain after sloughing and the diarrhea continues, the patient rapidly emaciating, and finally dying exhausted within a couple of months. Emaciation is very marked in these patients. Corneal ulceration may occur.

Stools.—As many as twenty to thirty may be passed in twenty-four hours, colicky pains usually precede them, and they are followed by straining and severe tenesmus. Movements at first are copious, later scanty, and chiefly mucus and blood. Intermissions and exacerbations of diarrhea occur, gangrenous dejecta, dark red brown and containing gray or black fragments of tissue (foul in odor), may be passed, or pure blood. Amebæ are present in the stools.

Abdominal Pains.—These are quite severe, chiefly before evacuation, and sometimes continuous. They are frequently located in the umbilical region and left iliac fossa; at times in the right iliac fossa, and may simulate appendicitis. Pressure increases the pain.

Tenesmus.—This consists of pressure and constriction in the rectum and a desire to go to stool. It may be continuous and accompanied by dysuria or strangury.

Other Symptoms.—Fever may occur, generally of moderate type, and it may be accompanied by chills at the onset. Temperature is irregular. Anorexia, nausea, and vomiting may occur. There may be severe prostration, cold extremities, delirium, stupor, drowsiness, and cerebral disorders.

Chronic Dysentery.—This type of dysentery shows several forms. It may be rather mild, characterized chiefly by diarrhea, with no blood or tenesmus, following on the mild type previously described. This may continue a number of years.

Dysentery in more marked cases is subacute from the onset or gradually passes into the chronic stage. There are generally alternating periods of diarrhea and constipation covering several years. During the exacerbations there are pain, passages of blood and mucus, tenesmus, and a slight rise of temperature. Many such cases do not feel especially ill between attacks and keep fairly well nourished. The appetite is often irregular in this type, and errors in diet are followed by exacerbations of the disease. The tongue is red, glazed, and beefy.

In more severe cases emaciation may be extreme and the patient be confined to bed most of the time. There are loss of appetite and nausea; diarrhea is quite persistent, there being mucus or mucus and blood in the stools, with attacks of colic and tenesmus. There may be some periods of improvement.

Infection in Children and in the Aged.—Musgrave reports cases in children from the age of six months to ten years in the Philippines. Children seem to present a natural immunity, he believes, and when infection does occur it seems of a mild type and readily yields to treatment.

The symptoms resemble those of the mild type in adults. In the aged there also seems to be a natural immunity, but when the disease was established it ran a severe and rapid course.

Liver abscess is infrequent in the very young and aged; Musgrave notes it is infrequent in the natives.

The only certain method of diagnosis is by microscopic examination of the feces or discharges and finding the amebæ.

Other parasites are found at times associated with the amebæ, such as the trichomomedæ, ova of uncinariæ, embryo strongyloides, tenia, oxyuris, etc.

Circulatory System.—The pulse may be of good quality at first; later, rapid and feeble. There are the changes of secondary anemia. Early, the blood is normal; later, the red cells are those of anemia. Still later, they may become irregular in size and shape and the count be reduced.

Leukocytes.—An increase in polynuclears and eosinophiles. Hemoglobin decreases with the red cells. Spleen usually not enlarged.

Temperature may be absent, moderate, or intermittent. In cases that are complicated, especially if there is diphtheritic inflammation of the colon, it may become quite high. With liver abscess temperature is frequent, but not constant. If there is mixed infection in the liver, bacteria with the amebæ, the temperature may be intermittent or remittent and resemble malaria or endocarditis. Sometimes it may be subnormal.

Nervous System.—Neuritis and neuralgias may be present.

Pain.—This varies in type and intensity.

Tenesmus.—More often is an indication of secondary involvement or complications. It is never present in the latent forms, and is often absent or very slight in the moderately severe cases.

In acute cases with diphtheritic process or secondary infection, as from Bacillus dysenteriæ (Shiga), it may be severe. It is more likely absent when the lesions are in the cecum or upper colon.

Colicky pains frequently occur, and at times severe colic.

The dull, aching *abdominal pain* is often prodromic and persists during the course of the disease, at times interfering with the patient's rest. The greatest intensity is usually along the colon, as demonstrated by palpation. Sometimes it is confined to the cecum. It may be complained of in the back. It is probably due to the ulcerative process in the bowel and, as demonstrated in the latent cases, as shown by post mortem, is the only indication during life of serious lesions. These pains are often the worst at night.

Burning pains ("heart-burn in the abdomen") Musgrave states may be general or local; when the disease is in the sigmoid or rectum, these pains may be intense, and extend down the backs of the thighs to the calves, and old cases of sciatica may be started up. Musgrave holds that the beginning of sciatica, associated with such cases, is of sufficiently frequent occurrence to be suggestive.

Appendicitis pains are at times stimulated, but examination will show that the tenderness is located in the caput coli. The differential method by Morris' point would be of value in these cases, as described under Appendicitis.

Neuralgia, myalgia, and arthralgia may be present. There is persistent dull headache in the back of the head and neck.

The genito-urinary, respiratory, and special senses are rarely involved, as is also true of the joints and osseous systems, though complications may occur.

Diagnosis.—In amebic dysentery there are so many clinical types that the diagnosis is by no means easy, and can only be made absolutely by microscopic examination of the feces.

In regions where the disease is endemic and the microscope is not available one can reasonably infer its presence by the most valuable symptom, to which I have already alluded, namely, "abdominal soreness which is increased on pressure and extends along the course of the colon, especially when there is maximum intensity over the cecum and ascending colon."

If bowel movements are present, their odor and the appearance of blood are of chief diagnostic import. Indigestion, pain, nausea, and other symptoms are not as important.

Often a thickened tender colon may be felt on palpation. These symptoms give the nearest approach to diagnosis without microscopic examination of the feces. Loss of weight is a guide to the infection.

Musgrave believes that the presence of amebæ in the stools in tropical regions should be considered diagnostic for purposes of treatment.

Amebiosis should be treated, even if other complications are present.

Prognosis.—The disease is generally milder in children and in the natives of the tropics. The course is shorter and the mortality higher among the aged. Previous good health is a favorable factor.

The *shorter the duration of the disease* and the *earlier the local treatment*, the *better is the prognosis.*

The higher up the lesion, the greater the mortality and the less active the clinical symptoms of dysentery. Infections of the cecum are the most serious.

Under proper treatment recovery is the rule in young and well-nourished adults if the disease is not of long duration. The early diagnosis and treatment are the important features, as otherwise the apparently mild cases may assume a dangerous character. Tendency to relapse or chronicity are characteristics. Abscess of the liver is a serious complication.

Treatment.—*Medical.*—The disease is contracted probably in the same way as typhoid fever, and the same prophylactic measures should be used. Infection through the drinking-water is undoubtedly the chief method.

Musgrave holds that the best rule to observe in countries where the disease is endemic is to "take nothing into the gastro-intestinal tract which has not been sterilized." He has found the amebæ in the drinking-water; on dishes washed in tap-water; in the soil from contamination; on the surface of uncooked vegetables, such as lettuce; on raw fruits; from hand contamination; and in ice-cream, water-ices, and milk.

Prophylaxis, when the disease is endemic or during epidemics, is very important.

All drinking-water should be boiled, and dishes should be washed in boiled water, also the hands.

Raw fruits and vegetables should first be placed on ice, and then have scalding water poured over them, which kills the amebæ. Ice-cream and water-ices should not be taken.

The vaginal douche and especially rectal enemata from tap-water should be avoided.

The stools should be disinfected in carbolic acid (1 : 20) or in bichlorid of mercury (1 : 1000), and the same precautions taken with linen—soaked in carbolic acid (1 : 20) and boiled.

Care of the hands and the prevention of fly infection (by screens) are necessary.

We have already noted that mixed infection with the Bacillus dysenteriæ (Shiga) may be present.

The *acid of the stomach lessens the chance of infection*, and *acid mixtures* may be given, such as *dilute hydrochloric acid.*

In *acute dysentery* the patient should be put to bed and placed on a liquid diet: barley-water, rice-water, bouillon, broths, gruels, white of raw egg, tea, also peptonized milk diluted with lime-water (at least 25 per cent. or more), or peptonized and diluted, or equal parts of milk and barley-water. With some sour milks agree, as kumyss, etc.

Sanatogen is an excellent preparation and somatose is of service. Personally I prefer milk-free diet and only the strained broths, gruels, etc., and 10 per cent. gelatin solution flavored with vanilla, ℥iv to vj (125–185 cc.). Give nourishment in divided doses (2 quarts daily) and a large amount of acidulated water. If the temperature is over 102.5 F., only water is given until defervescence to that point. Later the diet can be increased.

Hot applications or poultices should be employed over the abdomen for the relief of pain or colic.

Internal Medication.—Musgrave and Osler both object to the use of bismuth preparations for the diarrhea, on the ground that they coat the ulcers and interfere with their local treatment. I believe this probably to be true as regards bismuth subnitrate or subcarbonate, which would be required in large doses. I have occasionally employed bismuth subgallate, gr. 5 to 10 (0.3–0.6) t. i. d., in combination with other remedies, apparently with benefit.

On the other hand, I have seen at times that, in spite of all treatment, too frequent movements continue either in the acute or chronic cases. In such event I have employed bismuth subnitrate as much as gr. 90 to 120 (6.0–8.0) in divided doses in twenty-four hours with good results. This is preferable to the use of opiates, and I only employ it to avoid such. I have never had nitrate poisoning from large doses of bismuth subnitrate. It would seem a more likely occurrence in children.

In the initial stage the patient should be given magnesium or sodium sulphate, ʒj to ij (4.0–8.0), one or two doses, so as to thoroughly cleanse the bowels. Calomel, gr. 5 (0.3), may be given, or a single dose of castor oil, ℥iss (45.0).

In young persons these remedies in smaller doses.

Some recommend ♏xx (1.18) of laudanum, followed in half an hour by gr. 20 to 30 (1.3–2.0) of pulv. ipecac, after the saline treatment; but, like Musgrave, I believe this of no special service in these cases.

Occasionally salol, gr. 3 (0.194), with guaiacol carb., gr. 3 (0.194), and gr. 1 (0.065) of pulv. ipecac, given several (three or four) times a day, with small doses of Dover's powder, gr. 2 to 3 (0.13–0.194), have proved of service.

Strong has reported good results in some cases by giving internally acetozone (1 : 5000 or 1 : 3000) in carbonated water, 1 to 2 liters (quarts) in twenty-four hours in divided doses.

I have recently employed 1 liter (quart) of acetozone (1 : 1000), given in divided doses by mouth during the day with good results. Just before administering, the dose can be flavored with orange juice to make it more palatable.

Among valuable astringent remedies are:

Salicylate of guaiacol (guaiacol-salol), gr. 5 to 10 (0.3–0.6); tannalbin, gr. 10 (0.6); tannigen, gr. 10 (0.6); or tannoguaiaform, tannopin, and tannocol, given in doses of gr. 5 to 10 (0.3–0.6) three or four times a day.

Hydrochloric acid with pepsin or alone, or nitromuriatic acid are of value. Musgrave recommends hydrochloric acid. Thus:

℞. Acidi hydrochlor. dil. }
Comp. tinct. cinchona } .... āā ʒiij (12.0)
Aq. destil. .... q. s. ℥iv (125.0).—M.
Sig.—ʒj to ij (4.0–8.0) in water t. i. d. before food.

Vomiting should be treated by the methods described under Acute Gastritis. Small doses of Dover's powder may be required for persistent diarrhea.

Local Treatment.—This is of *extreme importance*, not only in the acute but also in the latent and chronic cases.

Extensive researches were conducted by J. B. Thomas, reported in Bulletin 32, Bureau Government Laboratory, Manila, who found the following solutions destructive to amebæ or inhibiting their growth:

Acetozone, 1 : 1000, most destructive to amebæ, and alphozone, 1 : 1000, nearly as much so.

They destroy other bacteria as well.

Protargol and argyrol, 1 : 500, were excellent antiseptics. Sulphate of quinin, 1 : 500, preferable strength, or bisulph-quinin; nitrate silver, 1 : 2000, of service; thymol, 1 : 2500, readily destroys amebæ; also permanganate of potash, 1 : 2000, is useful.

Hydrogen peroxid was recommended by Harris some years ago, and I have used it successfully for some time.

Cold water (under a temperature of 45° F.) has been highly advocated by J. P. Tuttle as destructive to the amebæ and also removing them from the bowel. He sometimes employs 5 to 10 per cent. hydrogen peroxid in this injection, and places the patients in the knee-elbow position and has them retain the injection for a considerable time (one-half hour).

A *glass irrigator*, attached to a colon-tube, with the opening preferably at the end, can be employed; if the ulcers extend low down into the rectum or there is extreme tenesmus, then an ordinary rectal tip.

The foot of the bed should be elevated 12 to 18 inches and the patient placed in the Sims position, or the hips can be elevated on a pan. The patient's position should be changed and he should be moved so the fluid will gravitate into the caput coli by movements described as "rotation method" under Enteroclysis.

Musgrave recommends the injection of at least 1 to 2 liters (quarts), which should be retained five to fifteen minutes, preferably the latter. At the commencement, if there is much irritation, often a smaller quantity must be used.

The knee-elbow position advised by Tuttle is excellent in the latent or chronic cases, but I would not advise it in acute conditions.

If there is much irritation one can precede the antiseptic injection one-half hour by a small enema of normal saline solution—℥ij (60.0) containing gr. ¼ (0.016) of morphin—or with tincture belladonna, ♏x (0.592), alone or together.

This should only be done once or twice in the first twenty-four hours and not repeated.

Musgrave has suggested taking advantage of the action of reversed peristalsis by giving occasionally a preliminary enema of a 7 to 8 per cent. salt solution, about 1 pint (500 cc.), containing gr. ¼ (0.016) morphin, one-half hour before the antiseptic injection.

He then employs acetozone (1 : 5000 to 1 : 3000), combined with quinin (1 : 1000 to 1 : 750), or gives the injections alternately, employing one to five enemata every twenty-four hours, according to the severity of the case.

Alphozone, same strength, or hydrogen peroxid (1 : 10) can be substituted.

We must remember that quinin affects some cases badly, causing gastric symptoms, vomiting, and headache. It must be omitted or weaker solutions given to such patients.

Quinin is much more efficacious in the strong solutions (1 : 1000 to even 1 : 500). The usual solutions suggested are much too weak. One can take advantage at the same time of the cold injections, as suggested by Tuttle, unless the patient have an idiosyncrasy to cold or renal complications. In such event a hot injection at 120° F. is of service.

An excellent method is to alternate, giving an enema of acetozone (1 : 1000), and the next enema of quinin (1 : 1000 to 1 : 500), cold, at 50° to 40° F.

If the quinin disagrees, then alternate the acetozone or alphozone with thymol (1 : 2500), or protargol or argyrol (1 : 500). The silver nitrate is at times found irritating in the acute cases.

Permanganate of potash (1 : 2000) is also of service, or hydrogen peroxid, ℥iv (125.0 cc.) to 1 liter (quart).

The local treatment is of chief importance to check the lesions and prevent liver abscess. In the latent cases the bowels should be opened freely with magnesium sulphate and daily injections of quinin and acetozone given. In cases of marked tenesmus, where the large or even small injections cannot be retained, recurrent irrigations with the antiseptic solutions, 1 or 2 gallons—1 pint (500 cc.) being kept in the bowels, with marked elevation of the bed and the solution of one-half strength—are of great service.

Chronic Dysentery.—In the *chronic cases* the diet should be quite liberal, but indigestible and *rich food* should be avoided. Mashed potatoes, boiled rice, and constipating food are often indicated in the diarrheal cases, and the avoidance of fruits and green vegetables. Milk, eggs (raw), the sour milks, and fats are of value. Judgment must be used in each case as to what will agree, whether liquid, semisolid, solid food, or combinations. Sanatogen is of value, and the iron preparations are often indicated. The tannic acid preparations, as suggested in the acute cases, should be employed. Occasionally large doses of bismuth subnitrate are necessary. Quinin and acetozone injections should always be employed; and nitrate of silver (1 : 3000 to 1 : 2000) is of value, used once or twice a week to heal the ulcers. Change of climate is valuable.

In all cases of fever, sponging (alcohol) is the best method of treatment. I deprecate the use of antipyrectics. If heart stimulants are required, small doses of strychnin, caffein, and spartein, or camphorated oil by hypodermic.

I have, moreover, a suggestion which I believe may prove of value. Piffard and Tousey have demonstrated that Morton's claims regarding the production of internal fluorescence by the *x*-rays after the administration of small doses of fluorescein or quinin are fallacious. However, the use of gr. ¼ (0.016) fluorescein in ℥vj (200 cc.) of water,[1] and placing the patient in the full electric-light bath for fifteen minutes or more, I believe, might prove of service in latent, chronic, or even in acute dysentery. There is great heat penetration from this light bath, as has been demonstrated in rheumatic and other conditions, and necessarily a certain penetration of light rays. The heat, light, and fluorescence are destructive to amebæ.

Musgrave demonstrated that violet light, *x*-ray light, and fluorescence inhibit amebic action.

Carbonic Acid Gas for Tenesmus.—Rose has demonstrated that the injection of CO_2 into the rectum will relieve tenesmus. It is worthy of use as an adjunct and can be given by his bottle.

Intestinal hemorrhage from dysenteric ulcers should be treated by high injections of 10 per cent. gelatin or, preferably, Trémoliére's solution, or, rather, a modification:

Trémoliére.—Gelatin, 5 per cent. solution, containing calcium chlorid, 2 per cent.

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[1] The fluorescein solution should be given by high enema; soda bicarbonate, gr. xv, should be added before injection, and water, q. s. 1 quart (liter).

Trêmolière (*Modified*).—Calcium lactate, gr. 20 (1.3); gelatin (10 per cent.), ℥viij (500 cc.).

Hot (120° F.) or cold (40° F.) astringent injections—ʒj (4.0) alum to 1 pint (500 cc.)—may be required. Avoid cold if there is shock.

Morphin, gr. $\frac{1}{8}$ to $\frac{1}{4}$ (0.008–0.016), by hypodermic at once; ernutin, ♏v (0.296), hypodermically, or fluidextract of ergot, ʒj (4.0), internally; ice-bag locally; calcium chlorid or lactate, gr. 10 (0.6), by mouth every three hours, gelatin solution (5 per cent.) may also be taken by mouth. Hypodermoclysis may be required if there is marked shock, or even infusion.

"*Surgical Treatment.*—In cases of chronic dysentery of long duration which do not respond to medical treatment, or in such cases with repeated acute exacerbations, surgical procedure is indicated. This is true of either the amebic or bacillary type of dysentery.

"*Appendicostomy.*—This operation was first suggested by Weir. It consists in suture of the appendix to the abdominal wall and skin, and removal of its apex. Subsequent irrigations of the large intestine are carried out by means of a small tube or catheter, which is inserted through the lumen of the appendix.

"*Cecostomy.*—An incision is made over the cecum and the latter brought into the abdominal wound. The cecum is incised and sutured to the abdominal wall. A catheter or drainage-tube is then inserted for the purpose of irrigation of the colon. Gibson has devised a valve operation, separating the abdominal muscles in a special manner so as to prevent leakage and ultimately secure a more ready healing. Cecostomy is indicated when the appendix is diseased or in an abnormal position, so that appendicostomy is impossible.

"*S. G. Gant's Modified Cecostomy.*—Gant[1] makes his incision over the cecum nearer the ileocecal junction. After opening the cecum and suturing it to the abdominal wall by means of a special director, he inserts a drainage-tube or catheter through the ileocecal valve into the ileum. A second tube is inserted into the cecum. In cases where ulceration of the ileum is present, it is thus possible to irrigate both ileum and colon."

Excellent results have been reported from these methods.

BACILLARY DYSENTERY

Definition.—A form of colitis, frequently an ileocolitis, usually of an acute type; occurring sporadically and in severe epidemics; attacking children as well as adults, and characterized by pain, tenesmus, and the frequent passage of blood and mucus; the result of infection by a specific bacillus, of which there are various strains.

Etiology.—Owing to improvement in sanitary conditions bacillary dysentery is less frequent. This is the type which has proved such a scourge, as epidemics in crowded asylums, institutions, and camps. It is one of the great camp diseases, and I have already referred to the fact that Woodward collected 259,071 acute cases during the Civil War. The disease prevails in the Philippines, Porto Rico, Cuba, and in South Africa. In Japan a fatal type has prevailed, especially in summer and autumn, having a mortality of over 25 per cent. In 1899 there were 125,489 cases with 26,709 deaths, collected by Eldridge. Most of the severe epidemics in the tropics are of the bacillary type, and the same form prevails in the temperate climates.

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[1] New York Med. Jour., Aug. 15, 1908.

Bacillus Dysenteriæ.—In 1892 Ogata, during an epidemic of dysentery in Japan, isolated fine bacilli which when introduced by the mouth or rectum produced ulceration in the intestines of cats. In 1897, during a severe epidemic, Shiga isolated the Bacillus dysenteriæ and described its special characteristics, demonstrating it to be the specific cause of the disease. Flexner and Barker found in the dysentery in the Philippines an identical organism, and Strong, Musgrave, and Craig have made a careful study of it.

It has been found in acute dysentery in Porto Rico. Out of 1328 cases of dysentery in Manila, Strong and Musgrave report 71 of the bacillary type, 51 suspected bacillary, and 561 amebic.

Kruse, in Germany, has isolated an identical bacillus. Vedder and Duval demonstrated that sporadic cases in adults in Philadelphia, and also epidemics in the Lancaster County Asylum, Pennsylvania, and in the Almshouse, New Haven, were due to the Bacillus dysenteriæ. Duval and Bassett, during the summer of 1902 at Mount Wilson Sanitarium, first demonstrated that certain forms of summer diarrhea in infants were due to Bacillus dysenteriæ, and under Flexner's direction at the Rockefeller Institute investigation, into the cause of infantile diarrhea in New York, Boston, Philadelphia, and Baltimore, showed the Bacillus dysenteriæ present in 63 per cent. out of 412 cases.

Several strains of this bacillus have been found, and the Flexner-Harris type is the one most frequent in the United States. These strains have been determined by the relative agglutinative power of immune serum upon the bacilli isolated, and also by the action of the bacilli upon various sugars. Flexner recognizes three types:[1]

1. "Shiga type" attacks glucose, without action on other sugars, including mannite and lactose.

2. "Flexner-Harris type" attacks glucose, mannite, and dextrin, not lactose.

3. "Bacillus" (Hiss and Russell) attacks glucose and mannite. No action on dextrin and lactose.

The lesions produced by the different strains are identical. The organism agglutinates with the blood-serum of cases with acute dysentery, as well as with the serum of immunized animals. The Flexner-Harris type agglutinates in dilutions of 1 : 1000 to 1 : 1500.

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[1] Hiss now recognizes four groups based on fermentative characteristics, and Shiga has added a fifth, intermediate between the acid and non-acid bacilli.

In two instances the organism has been isolated by Duval in the stools of healthy children. In *dysenteric stools it is most readily isolated from the particles of mucus.* The organism has not yet been isolated outside the human body, but the belief is that it is probably water-borne, and that the same prophylactic measures should be taken as in typhoid fever. Camp epidemics clearly originate in this way, and the care taken by the Japanese in regard to the latrines and water-supply practically eliminated the disease during their recent war.

Morbid Anatomy.—In acute cases, when death has occurred during the first week, the mucous membrane of the large intestine is swollen, hyperemic, of a deep red color, and presents elevated coarse ridges and folds. There are ecchymotic patches scattered throughout the swollen mucosa; over the surface there is usually a superficial necrotic layer, which can be brushed off lightly with the finger. This may be in patches or over large areas. There is no ulceration, but only the superficial general necrosis of the mucosa. They are, in effect, superficial erosions which give it a worm-eaten appearance. This superficial necrotic layer is, in effect, a fine pseudomembrane.

The solitary follicles are swollen and red, but their prominence is obscured in the involvement of the mucosa.

In severe cases the entire coats of the colon may be stiff and thick, and the mucous membrane greatly increased in thickness, grayish-black in color, extensively necrotic, and in places gangrenous. The submucosa is often enormously thickened and edematous. The serous surface is often deeply injected and the vessels of the mesentery, especially near the sigmoid and rectum, may be distended.

The ileum for 10 or 15 cm. is quite frequently involved (which is rare in amebic dysentery), having a deeply hemorrhagic mucosa with superficial necrosis. Peyer's patches and the solitary glands may be moderately swollen. The specific bacilli and various cocci[1] are abundant in the necrotic mucous membrane, and are said by Strong to be seen in all the coats.

In the subacute cases there is less thickening of the intestinal walls, there is less necrosis, the solitary follicles are more swollen, the mucosa less red, there are superficial erosions, and no ulcers. The disease, as suggested by the complications, is evidently characterized by a more or less acute *general toxemia*,[2] starting from a localized process.

Symptoms.—The incubation period is not more than forty-eight hours. The onset is usually sudden and characterized by fever, pain in the abdomen, and frequent stools, first containing mucus, and later consisting chiefly of mucus and blood. The movements increase in frequency and are associated with tenesmus, which becomes very marked. The movements may occur as frequently as every half-hour, and there is much straining. The tongue is coated with a white fur and there is excessive thirst; nausea and vomiting may occur. The abdomen is not distended, but there may be tenderness, especially over the colon. There are cramp-like pains in the abdomen. The spleen is not usually enlarged. The temperature rises to 103° or 104° F. It may run an irregular course and rise or fall before death. The pulse increases in rapidity (100 to 120 or even to 150) and becomes rapid and feeble. Urine is decreased and may contain albumin. Liver not enlarged and *no liver abscess;* moderate leukocytosis may be present. In very acute cases the patient becomes seriously ill within forty-eight hours, the movements increase in frequency, the pain is of great intensity, severe headache, and the patient becomes delirious and dies on the third or fourth day. Lobar pneumonia, bronchopneumonia, acute bronchitis, and fibrinopurulent pleurisy may occur.

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[1] Streptococci and various other types may be present.

[2] The toxins of dysentery are probably excreted by the bile and also through the intestinal mucosa, thus aiding in the damage to the intestines. Probably the toxins are responsible for cerebrospinal lesions (Herter).

In cases of moderate severity the symptoms abate, stools lessen, temperature falls, and within two or three weeks the patient is convalescent. In the subacute cases the attack may last many weeks or even months, the patients have three to five bloody mucous stools in twenty-four hours, and become very emaciated. One of Strong's cases died on the sixty-fourth day. The Bacillus dysenteriæ is found in the stools and agglutinates with blood-serum.

Other Clinical Types.—The description just given applies to the types of bacillary dysentery such as seen in Japan, the Philippines, and the tropics, and the features of that in adults in temperate climates differ in no essential, except in many cases it is less severe.

Duval, as noted, has found one of the bacillary dysentery strains in sporadic cases in Philadelphia and elsewhere, and the probability is that most cases of non-amebic dysentery belong to this type.

The so-called acute catarrhal dysentery is unquestionably a sporadic form due to the Bacillus dysenteriæ. This is the more probable when we consider that in ileocolitis (dysentery) in infants we find a catarrhal type. In infants there are four types of lesion found on autopsy (Holt):

Follicular ulceration; catarrhal inflammation; catarrhal inflammation with superficial ulceration; membranous inflammation.

This last differs from the membranous type in adults, in that there is little psuedomembrane and no deep sloughing. Holt has well described these varieties.

Diphtheritic[1] dysentery is a type of the bacillary form with great necrosis and infiltration of the mucosa. It is believed that other types of bacteria are also often associated in the process.

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[1] The pure dysentery bacillus, unlike the typhoid bacillus, does not lead to bacillemia or bacilluria (Herter, Bacterial Infections of the Digestive Tract). In the diphtheritic type, a mixed infection in which streptococci undoubtedly are prominent, the condition is unquestionably, in my opinion, a *general infection*, as shown by the complications.

The secondary diphtheritic dysentery is a common terminal event in many acute and chronic diseases. Vedder and Duval have demonstrated that the bacilli are present in these cases.

Peritonitis is rare, either through extension or by perforation. When it occurs about the cecal region, perityphlitis results, or when low down, periproctitis.

In 108 cases collected by Woodward, perforation occurred in 11.

Abscess of the liver is *very rare*. A few cases occurred in the Civil or South African War. In the tropics malaria and dysentery may coexist, and typhoid and dysentery coexisted quite frequently in the Civil War. In ordinary practice it is very rare.

Complications.—Acute bronchitis, pneumonia, bronchopneumonia, pleurisy, gangrene of the lung, albuminuria, meningitis, paralysis, paraplegia, in many cases due to a neuritis, are not uncommon; thrombosis of cerebral sinuses, embolism (cerebral), rheumatic pains and swollen joints (analogous to gonorrheal arthritis), pericarditis, endocarditis, periproctitis, perityphlitis, and occasionally pyemic manifestations, such as pylephlebitis or abscess of the spleen, may occur. Anemic edema may be present in protracted cases. Chronic Bright's disease is an occasional sequel and intestinal stricture is *rare*. Persistent dyspepsia and irritability of the bowels may follow.

Prognosis.—In the very severe cases the prognosis is bad, the patient often dying within one to two weeks. This is especially true in epidemics. In milder cases convalescence begins by the end of the second week. Chronic cases may run weeks or even months, the patient becoming very emaciated. They may die at the end of several months. Convalesence is slow in any but the mild cases.

Treatment.—*Prophylaxis.*—The same precautions should be exercised as regards boiling drinking-water, avoiding green vegetables, cleanliness of the hands, destruction of flies, disinfection of stools and linen, as are carried out in amebic dysentery and in typhoid fever.

Medication.—The bowels should be at once thoroughly cleared with castor oil, ℥j to ij (30.0–60.0), or with magnesium sulphate or sodium sulphate, ʒj to ij (4.0–8.0). Buchanan has had excellent results by the sodium sulphate treatment; he gives ʒj (4.0) sodium sulphate four to even eight times a day for two to three days until blood and mucus have disappeared. He has treated 855 cases with 9 deaths—an excellent record.

The old ipecac treatment is still in considerable repute in tropical countries. No food is taken for three hours, then 20 drops of laudanum are administered and one-half hour later gr. 20 to 60 (1.3–4.0) of pulv. ipecac. If this is vomited, the dose is repeated in a few hours.

In South Africa the saline treatment was often given combined later with the ipecac, and Washbourne reported good results.

Ringer recommends small doses of bichlorid, gr. $\frac{1}{100}$ (0.00065), every two to three hours, and large doses of bismuth subnitrate have been used with success, at least ʒss (2.0) and sometimes as much as ʒj (4.0) every two hours daily for the first few days. The following I have found the most rational method of internal medication.[1]

The bowels should first be thoroughly cleared by the administration of ℥iss to ij (45.0–60.0) of castor oil in the adult, or by several doses ʒj to ij (4.0–8.0) of magnesia sulphate or sodium sulphate; calomel, gr. v (0.3), may precede this. A hot application should meanwhile be applied to the abdomen to relieve the pain, and if the latter be unendurable and there is considerable collapse, a very small hypodermic of morphin may be given, sufficient to render it endurable and not large enough to interfere with peristalsis. It is preferable not to administer opiates until thorough cleansing of the bowels has been accomplished. In fact, it is my belief that opium is given too frequently and in too large doses, so that material accumulates in the bowel which had better be expelled.

Bismuth subnitrate, which it is preferable, as a rule, not to use in the undermined ulcers of amebic dysentery, in this type is of great value. The dose should be large. After thorough clearing of the bowels, bismuth subnitrate alone or combined with equal parts of saccharated pepsin should be given.

At least gr. 20 to 30 (1.3–2.0) of this mixture or even gr. 20 (2.6) of bismuth subnitrate every two to three hours. Bismuth subcarbonate, gr. 10 to 15 (0.6–1.0), can be substituted. Bismuth subgallate, gr. 5 to 10 (0.3–0.6) t. i. d., or bismuth salicylate, gr. 10 (0.6) t. i. d., can be substituted in place of some of the usual doses of the other preparations; thus, for example, bismuth subgallate and bismuth subnitrate could alternate. Bismuth subnitrate, gr. 30 (2.0), mistura cretæ, ʒij (8.0), is an excellent combination in one dose, given every three or four hours. The tannin preparations, such as were suggested in amebic dysentery, can be given, three doses substituted for three of the bismuth in the course of twenty-four hours. Thus, if seven doses of bismuth were given in twenty-four hours, give three of tannin and four of bismuth. If the pain is severe, small doses of opium may have to be given, with the precautions noted. Children should receive proportionately small doses of all remedies. The opiates are described under Diarrhea.

Tenesmus should be relieved first by thorough bowel irrigation, and after this, if it continue, by inflation with a small amount of CO_2 (Rose's method), or by a small injection of starch-water and laudanum, ♏xv (0.888), with tincture of belladonna, ♏x (0.59). Opium should never be given first, thus tying up the foul secretions.

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[1] Urotropin, I believe, might be of service; urotropin, gr. v to x (0.3–0.6), combined with equal quantities of sodium benzoate, given three or four times daily. It might lessen the toxemia and be of service in the cases of mixed infection.

Strychnin, nitroglycerin, or camphorated oil by hypodermic may be required. Local treatment is of paramount importance. Zinc, sulphocarbolate of zinc, and silver nitrate are preferable in the chronic cases. Hypodermoclysis, or even infusion, may be required in shock or severe sepsis.

If there is much tenesmus, a preliminary suppository of belladonna ext., gr. ⅓ (0.022), alone or, rarely, combined with gr. ⅛ (0.008) cocain, or the injection of gr. ⅛ (0.008) cocain in ℥j (30.0) water, may rarely be necessary. The best method is to insert a recurrent tube and gently irrigate with normal saline solution at 110° to 115° F., or cold at 40° to 50° F., and then, forcing the tube in still further, continue the antiseptic irrigation.

I have found the recurrent method of irrigation with a double tube (preferably) or two tubes (soft catheters) of great value in these cases, the patient's hips or foot of the bed being elevated. The patient is gradually accustomed to the fluid, and the quantity allowed to run in before permitting return can slowly be increased and there is not the endeavor to expel the solution. A single injection of any size can often not be given.

On the other hand, 1 to 1½ liters (quarts) can be injected by the method of Musgrave in amebic dysentery in some cases.

In the *acute cases* acetozone, alphozone, hydrogen peroxid, and permanganate of potash are of especial value, in the same strength noted below as employed in amebic dysentery.

Delafield has secured excellent results by the use of bichlorid of mercury (1: 10,000), using 2 quarts (liters), but cautiously, with my recurrent tube. I have had good results by this method, but it should never be given by ordinary enema.

Acetozone (1: 1000), hydrogen peroxid (1 : 10), alphozone (1: 1000), and permanganate of potash (1: 1000) are excellent.

Mucol, ʒj (4.0) to 1 quart (liter) or even stronger, has been employed. This preparation contains the chlorid, borate, bicarbonate, and benzoate of soda, with zinc sulphocarbolate and essential oils.

Several irrigations or enemata may be necessary daily, depending on the tenesmus or character of the stool, one or two antiseptic, and the rest of normal saline solution, or flaxseed or gum-arabic.

If mucus and blood increase, a cathartic is again indicated, and I believe it should be given every three days in any event.

Carbolic acid should never be employed for irrigation.

Chronic Dysentery.—Bismuth is of value. Nitrate of silver injections are of service—10 to 30 gr. (0.6–2.0) to 1 pint (500 cc.)—and inject 2 to 3 pints if possible; if there is irritation, follow it with an injection of normal saline solution.

Protargol or argyrol (1: 500) are useful, and often preferable to silver nitrate.

Normal saline or normal saline with oil of peppermint, ♏x (0.59), can be used between the antiseptic injections, or an injection of flaxseed tea or gum-arabic solution. The latter are soothing.

Diet.—*Acute Cases.*—Fluid diet, broths, gruels, barley- and rice-water, beef juice, sanatogen, egg-albumen, and milk well diluted. Somatose is of use. Personally I prefer milk-free diet.

In the *chronic cases* fruits and green vegetables should be avoided and constipating food given if there is diarrhea. The weight of the patient should be increased by administration of fats. The *general diet rules* should be similar *to those in amebic dysentery.*

Serum Therapy.—Shiga immunized horses and produced a polyvalent serum from which he claims to have reduced the mortality of dysentery in Japan from 35 to 9 per cent.

Flexner has prepared a serum, but Holt states results in children have proved disappointing.

Complications should receive appropriate treatment.

CHAPTER XXV

TYPHOID FEVER

A MERE outline of this disease will be given for the purpose of differential diagnosis. Typhoid may be defined as a general infection caused by the Bacillus typhosus, characterized anatomically by hyperplasia and ulceration of the intestinal lymph-follicles, swelling of the mesenteric glands and spleen, and by parenchymatous changes in the other organs, such as in the kidneys, liver, etc. Clinically the disease is marked by fever, rose-colored eruption, abdominal tenderness, tympanites, diarrhea, and splenic enlargement, but these symptoms are inconstant.

History.—Louis, in 1829, gave the name to the fever. Gerhard, in 1837, first clinically differentiated between typhoid and typhus.

Etiology.—General prevalence. It prevails in temperate climates. Imperfect sewage and contaminated water-supply favor the distribution of the bacilli; filth, overcrowding, and bad ventilation aid in lowering the resistance of the individual. *Fingers, food,* and *flies* spread the disease from the infected person. It is prevalent in England, Wales, India, parts of Germany, and in the United States, where it occupies about fourth place in the mortality list. It has been one of the great scourges of armies, more destructive than powder and shot. The mortality in the South African and Spanish-American wars was greater from typhoid than from bullet wounds; among the American troops, about one-fifth of the soldiers in the national encampments had this disease. The former inefficiency of our sanitary methods was a blot upon our government. In great contrast to this were the remarkable results secured by the Japanese in the recent Russo-Japanese War, where, in comparison with our own records, typhoid and dysentery were practically negligible factors.

Season.—The disease is more prevalent in the autumn, though frequent cases occur during August.

Sex.—Both sexes are equally liable, but males are more frequently admitted to the hospitals.

Age.—Typhoid is a disease of youth and early adult life, the greatest susceptibility being between the ages of fifteen and thirty. Cases are rare over sixty. It is not infrequent in children. Infants are rarely attacked.

Immunity.—Not all exposed take the disease. One attack usually protects; two attacks have been described, and occasionally three.

Bacillus Typhosus.—The researches of Eberth, Koch, and others have shown that the disease is due to a special micro-organism. It is a short, thick, flagellated, motile bacillus with rounded ends, in one

There is hyperemia of the follicles; later an increase and accumulation of the cells of the lymph-tissue which may infiltrate the adjacent mucosa and muscularis; the blood-vessels are compressed, which gives a white anemic appearance to the follicles.

This process reaches its height from the eighth to the tenth day, and then undergoes either resolution or necrosis.

2. Necrosis. When the hyperplasia is marked, resolution is no longer possible. The blood-vessels become choked; there is a condition of anemia; then necrosis and sloughs form, which must be separated and thrown off. This process is always more intense toward the ileocecal valve. The necrosis is variable, it may pass deep into the muscular coat, and even perforate the peritoneum.

3. Ulceration. Sloughing is effected from the edges inward, and results in the formation of an ulcer, the extent of which is directly proportionate to the amount of necrosis. The muscularis usually forms the floor of the ulcer.

4. Healing. The mucosa extends from the edge and a new growth of epithelium is formed, as are the glandular elements. The healed ulcer is depressed. Healing is never associated with stricture.

Large Intestine.—The cecum and colon are affected in about one-third of the cases, and the solitary glands are sometimes enlarged.

Perforation.—About one-third of the deaths, Scott's statistics state, are due to perforation. It occurred in 3.6 per cent. of all cases.

The German statistics are much lower; in Munich only 5.7 per cent. of deaths are due to perforation. Among 1500 cases at the Johns Hopkins Hospital there were 43 of perforation; 20 were operated on, and 7 of these recovered. The site of the perforation is usually in the ileum, within 12 inches of the ileocecal valve. It may be from a pin-point to large size.

Death from Hemorrhage.—This occurred in 12 of 137 deaths in Osler's 1500 cases. He could not find the bleeding vessels.

Mesenteric glands are hyperemic and swollen. Necrosis is common; abscesses may occur, causing peritonitis or hemorrhages.

Spleen enlarged, infarction not infrequent. Rupture may occur.

Bone-marrow.—Same changes as in lymphoid tissue, and there may be foci of necrosis.

Liver.—Parenchymatous degeneration. Liver abscess has been found, also acute yellow atrophy. Pylephlebitis may occur.

Gall-bladder.—Acute cholecystitis may be present.

Kidneys.—Cloudy swelling, with granular degeneration; less commonly an acute nephritis; miliary abscesses or diphtheritic inflammation of the pelvis may occur; also infection by colon bacilli. With colon bacilli infection, chills, rise of temperature, and acute renal symptoms occur (W. H. Thomson).

Bladder.—Cystitis or diphtheritic inflammation. Orchitis and acute mastitis are occasionally met with.

Respiratory Organs.—Ulcer of the larynx; edema of the glottis;

diphtheritic inflammation; bronchitis; pneumonia; hypostasis; pleurisy; gangrene; abscess of the lung; hemorrhagic infarction and empyema may complicate.

Circulatory Changes.—Endocarditis, pericarditis, and myocarditis, endarteritis, arthritis of a peripheral vessel with thrombus formation may occur. Venous thrombosis is more frequent, especially of the left femoral.

Nervous Symptoms.—Meningitis is rare; optic neuritis may occur; the cause of aphasia seen in children is not positively known. Parenchymatous changes may occur in the peripheral nerves.

Voluntary Muscles.—The muscular substance, especially of the recti, pectorals, and adductors of the thigh, may undergo granular degeneration or hyaline transformation. Rupture, hemorrhage, or abscess have been found.

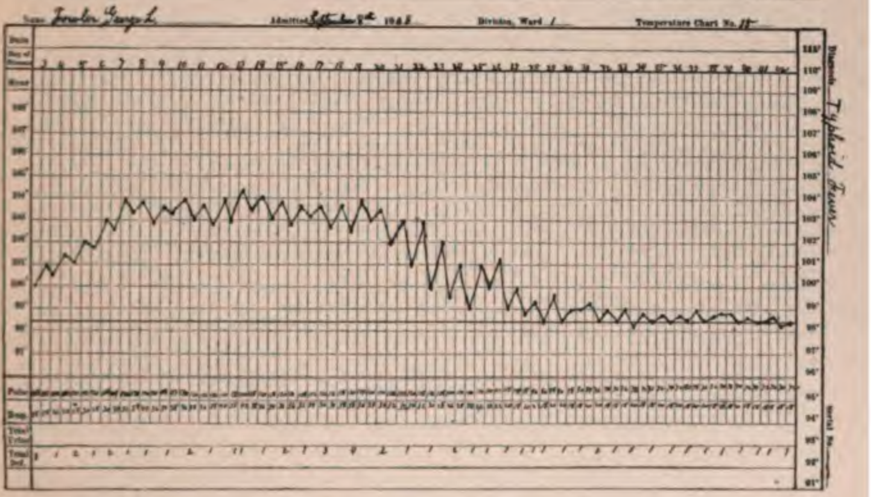

Fig. 209.—Typhoid chart.

Symptoms.—Incubation, eight to fourteen days, occasionally to twenty-three days, during which period there is lassitude and inaptitude for work. Onset is rarely abrupt, with occasional chills, There may be cough (bronchitis), epistaxis, headache, anorexia. diarrhea in many cases, but frequently early constipation, abdominal pain, and distention; and in some cases there is pain in the right iliac fossa. At the onset the patient usually takes to his bed.

During the first week there is in many cases a steady rise of temperature, the evening record rising a degree or more each day, and reaching 103° to 104° F. (Fig. 209). Variations of temperature are common. It may rise suddenly to 104° F. or may fall suddenly. It may be high A. M. and lower P. M.

The pulse is rapid, 100 to 110, full, but of low tension, and often dicrotic. The tongue is coated and white, the abdomen slightly distended and tender. Unless there is high temperature there is no delirium. The patient complains of headache and there may be mental confusion at night. The bowels are loose or may be constipated. At the end of the week the spleen becomes enlarged and the eruption appears in the form of rose-colored spots, seen first on the abdomen. They are raised, flattened papules, can be felt by the finger, and disappear on pressure. They come out in crops and may appear on the trunk or extremities. Desquamation may occur.

In the second week the symptoms become aggravated; the fever remains high and the morning remissions are slight, the pulse is rapid and loses its dicrotism. There are mental torpor and dulness; the lips are dry and the tongue may become dry. Tympanites, tenderness, and diarrhea, if present, become aggravated. The stools are described as pea-soup. Death may occur at the end of this week from hemorrhage or perforation, but generally later. In the third week the pulse ranges from 110 to 130. The temperature shows marked morning remissions and there is a gradual decline in the fever. Loss of flesh is more marked and weakness is pronounced.

Diarrhea and meteorism may in some cases occur for the first time. Unfavorable symptoms are pulmonary complications, feebleness of the heart, delirium with muscular tremor, and acute tympanites. The tongue may become brown or brownish black and the lips and teeth be covered with sordes.

Special dangers are perforation and hemorrhage.

The fourth week convalescence begins. Temperature gradually becomes normal; diarrhea stops; tongue cleans; the desire for food returns. In severe cases the fourth and fifth weeks present an aggravated picture of the third, the patient grows weaker, pulse more rapid and feeble, tongue dry, and abdomen distended. He lies in profound stupor with low muttering delirium and subsultus tendinum and passes the feces and urine involuntarily. Heart failure and secondary complications are the chief dangers.

In the fifth and sixth weeks protracted cases show irregular fever, and convalescence may not set in until the fortieth day or longer. During this period, recrudescence of temperature may occur from errors in diet, constipation, or excitement; or a relapse (re-infection) mav occur. With relapse there is a repetition of the ascent of the original fever. A rise in temperature from a complication usually has leukocytosis associated.

Modes of Onset.—As a rule, the *onset is insidious*. The following deviations may occur: Marked nervous symptoms, such as headache or cerebrospinal symptoms, with retraction of the head and convulsions; or mania; or stupor. Pulmonary symptoms, such as bronchitis, pneumonia, or pleurisy. Intense gastro-intestinal symptoms, with pain and vomiting, suggestive of poisoning, or in some cases simulating appendicitis. Acute nephritis is the first symptom. Ambulatory form, in which the patient keeps about and attempts to work. This runs a severe course and often a fatal issue results. Chills may occur at the onset and may be followed by sweats, and they are present with complications. Variations from the typic temperature are common, the step-like ascent does not always occur, but the fever may rise suddenly. A sudden fall of temperature is suggestive of hemorrhage before blood appears in the stools. Peliomata, maculæ cerulæ—pale-blue or steel-gray spots—are sometimes present due to lice. Erythema may occur. Bed-sores are not uncommon and boils are a troublesome sequel.

Blood.—Leukopenia (hypoleukocytosis) is present; lymphocytes are relatively increased. *Eosinophiles disappear* or are markedly diminished. The reappearance or increase of eosinophilia is a good prognostic sign. Leukopenia and absence of eosinophiles aid diagnosis. Hemoglobin and red corpuscles are reduced.

Severe meteorism is a danger-signal and predisposes to hemorrhage or perforation. Acute gastro-intestinal dilatation or acute ectasia alone may occur. Acute distention may even simulate perforation; and it is only possible to differentiate by relieving the distention by enteroclysis and lavage.

Symptoms of Perforation.—Sudden sharp pain, at times paroxysmal, often in the hypogastric region to the right of the median line; tenderness, sudden distention, and muscular rigidity. *This last is an important symptom.* There are shock, fall of temperature, pallor, sweating, and the Hippocratic facies. The temperature then rises, pulse rapid and feeble, respiration increases. Vomiting is often present. Leukocytosis and especially increase in the polynuclears. Percussion may show a flat note in the flank, due to exudate. Obliteration of liver flatness may be caused by tympany.

Abscess of the liver and cholecystitis may complicate. Gallstones in many cases are probably associated with the presence of typhoid bacilli in the gall-bladder. Under Lesions most of the complications are referred to. Loss of hair may occur.

Local neuritis, as in the arms, legs, or toes (tender toes), may occur. Multiple neuritis is a complication of convalescence. Poliomyelitis, tetany, and hemiplegia have been reported. Typhoid pyschoses may occur, also eye and ear complications, and retention of urine. Posttyphoid anemia may be severe.

Ehrlich's Diazo-reaction.—This test is not absolutely diagnostic, as it occurs in miliary tuberculosis, in malaria, and occasionally in other acute disease, associated with high fever. It is of accessory value taken with other data. Bacilluria occurs in about one-third of the cases. Acute appendicitis may complicate.

Osseous System.—Periostitis, osteitis, caries, and necrosis are troublesome sequelæ of typhoid, as are arthritis and typhoid spine. Colitis, simple catarrhal or of a septic (diphtheritic) type, may complicate.

Posttyphoid septicemia and pyemia are not uncommon. Furuncles, abscesses, and infarcts in various regions may occur. With children typhoid fever often runs a mild and irregular course.

Diagnosis.—The type of temperature, splenic enlargement, eruption, absence of leukocytosis, disappearance of the eosinophiles, together with Ehrlich's reaction, are the chief signs. Isolation of the typhoid bacilli from the blood, stools, and urine is diagnostic.

Widal's reaction is of positive value when found. It is generally not in evidence until the seventh to tenth day, sometimes not until convalescence, and occasionally not at all. Cabot claims over 90 per cent. reactions before the eighth day. Fortunately it is present in about 90 per cent. of cases. Blood examination will differentiate between typhoid and malaria. Ulcerative endocarditis has been mistaken for typhoid, but the presence of the heart lesion and the streptococci in the blood are diagnostic. With acute miliary tuberculosis the temperature is irregular or intermittent.[1] Respiration is more rapid and there is more cyanosis. Widal is negative, but leukocytosis is common. With typhoid we have the Widal reaction; there is leukopenia, splenic enlargement is present, and the typic eruption. Intestinal grip may for a few days simulate typhoid, as may intestinal toxemia.[2] The subsequent course, absence of Widal reaction, etc., clear the diagnosis.

Prognosis.—Mortality. Death-rate is variable, depending on the severity of the epidemic and when treatment has begun. Of recent years it has been from 5 to 20 per cent. Fat people stand typhoid badly. Meteorism, hemorrhage, high fever, and nervous symptoms give a bad prognosis.

Prophylaxis.—Care in drainage and water-supply are most important. Raw milk, raw oysters, and uncooked fruit and vegetables should be avoided during an epidemic. Raw oysters in New York should generally be avoided, unless the source of the supply is known. The urine, stools, and sputum should be disinfected. The sputum can be collected in cloths and burned, and bichlorid (1 : 1000) or carbolic (1 : 20) used to disinfect the other discharges. About twice the volume of the disinfectant should be mixed with the stools, and it should be left to stand for two hours before emptying. For disinfection of the bath water, ½ pound of chlorid of lime will render an ordinary bath of 200 liters sterile in one-half hour (Babucke). The nurse should wear a rubber apron and should wash it frequently with carbolic or bichlorid. When giving baths, rubber gloves, or especially *rubber finger-tips*, should be worn, and the hands thoroughly disinfected later. All bedding and the patient's night dress should be soaked two hours in 1 : 20 carbolic and then boiled. A special cup, dish, spoon, etc., should be used for the patient and disinfected on each occasion after using. It is best to keep them in the room and transfer the food from another vessel to the special cup or dish; if in a ward, after each feeding they should be placed in carbolic (1 : 20).

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[1] Tubercle bacilli may be found in the fluid by lumbar puncture, and there is an absence of typhoid bacilli in the blood-culture. The tuberculin test (ocular) or by injection is of value.

[2] The paratyphoid bacilli A. and B. may produce symptoms like a mild typhoid, and paratyphoid B. may be productive of meat-poisoning. The Bacillus fæcalis alcaligenes and Bacillus enteritidis (Gärtner) may incite symptoms simulating mild typhoid (Herter). These bacilli differ from the typhoid bacilli in agglutination reactions, etc.

After recovery, the room should be disinfected. Osler shows that 1.81 per cent. of cases of typhoid at the Johns Hopkins have been of hospital origin. I have noted great carelessness in our hospitals as regards fly protection, seldom observing screening of the patient during the fly season. Typhoid cases should preferably be kept in a special ward to avoid the danger of infection of other patients through carelessness. Mabon demonstrated this at the Manhattan State Hospital.

Chronic Typhoid Distribution.—Many cases are discharged from the hospitals while there are still typhoid bacilli in the urine and stools. Examinations of these discharges should be made.

Urotropin, gr. 5 to 10 (0.3–0.6), with an equal amount of sodium benzoate t. i. d. is excellent to disinfect the urine.

An interesting case of typhoid carrier of nearly seven years' duration, with five small epidemics to her credit, is reported by George A. Soper.[1] Wright has introduced a method of vaccination against typhoid which proved of considerable value in South Africa.

Treatment.—*General Management.*—The patient should be in a light, well-ventilated room, confined to bed. This should be single, with a comfortable mattress, covered with a blanket, and a rubber cloth placed under the sheet. Nursing and diet are the essentials.

Diet.—There is a tendency among many practitioners to endeavor to increase the resisting power to typhoid by increased feeding, and who hold that for this purpose the patient should lose as little weight as possible. In some cases solid food has been administered; in others liquids, with considerable dextrose and cream.[2] *It has been thoroughly demonstrated* that no matter what the intake, the nitrogenous output in this disease is always markedly in excess.[3]

Typhoid is an *acute general infection*, and in such conditions all the digestive functions are abnormal. There is change in the character of the bile. Stölmkow[4] noted disturbances in the pancreatic juice; and I recently referred to the diminution[5] of motor power and the lessening or absence of hydrochloric acid during the high temperature of typhoid fever. The parenchymatous changes in the liver and kidneys interfere with elimination by these organs, and the associated intestinal catarrh causes further interference with the digestive functions. Seibert, in 1889, noted that temperature and tympanites were lessened when milk was withheld, the fever falling to 99° or 100° F. on the ninth to twelfth days. Rectal irrigations he found of great value. He[1] gives rectal injections with 3 pints of warm normal saline solution, preferably several times a day, depending on the case, thus irrigating the bowel. During the first day of treatment cold water only is given as food. From the second day on, ½ pint of strained rice, oatmeal or barley soup, containing the extract of ½ pound of meat and the yelk of a fresh egg, well spiced, are given every three hours, five times daily. From the fourth day on, strained pea, lentil, potato, and tomato soup with rice, were added to the menu. Two or three zwiebacks were given with the soup at the end of the first week. Orange juice was given in water three times daily. Egg-albumen was not given on account of the probability of forming toxins. Before each meal 15 to 25 drops of hydrochloric acid were administered in ½ ounce of water. No alcohol was given except to topers, and camphorated oil was employed by hypodermic if stimulation was required. Cold baths were never employed, even in hyperpyrexia. Sponging was added if necessary. Opium was only used in bowel hemorrhage.

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[1] Journal of American Medical Association, June 15, 1907.

[2] W. Coleman (Jour. Am. Med. Assoc., Oct 9, 1909) advocates milk, cream, milk-sugar, and eggs, giving from 4000 to 5500 calories per day, and reports good results. The author disagrees with a method which places upon diseased organs twice the labor which they perform in health, with the dangers incident to excessive feeding.

[3] Finkler and Lichtenfeld, Centralblatt für die Allgemeine Gesundheitspflege, 1902.

[4] Pflüger, Archiv. Physiologie.

[5] Medical Record, June 20, 1908, and American Medicine, May, 1909.

Lesser[2] has treated all fevers above 102.5° F. with water alone, and below this point with broths, rice- and barley-water, also employing enteroclysis. Good results were secured by these methods in typhoid during the Spanish-American War.

The author's method is as follows: As gelatin[3] lessens nitrogen excretion, and as it aids in preserving weight, and furthermore causes no putrefaction in the intestines, it is of some value as a food. It also lessens tendency to hemorrhage.

Approximately, ℥iss (48.0) of gelatin in ℥xij (375 cc.) of water, gives a 12 per cent. solution. This gelatin solution can be slightly flavored with lemon, vanilla, or with a pinch of sugar, and be given in divided doses. Strained rice, barley, oatmeal, and chicken broths, the sum total not over 1 quart (liter), are also to be given in divided doses every three to four hours, the last feeding no later than 9 P.M. This makes in all about 1½ quarts (liters) of nourishment. The various foods are alternated for variety. The patient is not urged to eat. Whenever the temperature reaches 102.5° F. or more nothing but water is administered until it falls to that point. The *juice of several oranges* is given during the day. When the tempe ature falls to 99.5° F., the gruels are thickened and the yelks of 4 raw eggs added.

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[1] Medical Record, June 20, 1908.

[2] Ibid., October 19, 1905.

[3] The ingestion of 7.5 per cent. of the total heat requirement of the organism in the form of gelatin spares 23 per cent. of the body's proteid. Thus, in a total of 2800 calories required by a man of 154 pounds (Chittenden), 210 calories gelatin are necessary; 1 gm. of gelatin contains 4.1 calories, so about 50 gm. gelatin are required, or ℥iss (48.0). The gelatin and cereal gruels approximate 2000 calories, all that can be digested properly.

At least 1½ to 2½ quarts (liters) of water, to which dilute sulphuric acid, ♏xx (1.184 cc.), is added, are to be drunk by the patient during the twenty-four hours. Dilute nitromuriatic or dilute hydrochloric acid may be substituted.

For *the advocates* of milk, I would state that the *sour milks*, such as matzoon, bacillac, kumyss, and lactone-buttermilk, are preferable to plain milk. Effervescence should be allowed to a great extent to pass off from kumyss before administering, and it is preferable to dilute it some with lime-water or water. Matzoon and the thicker sour milks should be diluted one-half with plain water, or Vichy that has become flat, to avoid distention. Milk[1] if administered should be diluted one-half, preferably with barley-water or rice gruel. About 1½ quarts (liters) of milk or sour milks thus diluted could be given in twenty-four hours. I do not advocate their use. Carbonated waters while effervescing add to distention. Sanatogen, as much as ℥j (32.0) in divided doses, is of value, given in the broths. The bowels are freely opened by calomel, gr. 5 (0.3), or castor oil, ℥iss (45.0), on the first day, and thereafter hot saline enemata, 1½ liters (1500 cc.), at 110° to 115° F., or if gas, enteroclysis (recurrent), 1 gallon is given A. M. and P. M. as routine. Hemorrhage, perforation, or appendicitis are the only contraindications. There is one exception to this rule: gentle bowel irrigation with a tube and funnel, with hot normal saline solution at 120° F. during hemorrhage, lessens tympanites and helps contract the vessels. Performed *by the physician*, if guarded by a hypodermic of morphin, gr. ¼ (0.016), to prevent subsequent peristalsis, I believe, with Kaufmann, the procedure to be of value.

Sponging.—With diet and irrigation of the bowels *tub-baths* are rarely necessary. Cold sponging with alcohol and water, combined with friction when the temperature is over 102.5° F., generally suffices.

Baths.—I am not opposed to the Brand method as a scientific procedure for its additional effects on the pulse and on elimination. If the friction bath is given, it should be started at about 90° F., never given below 70° F. Often the warm bath is preferable.

Strychnin, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021), or Hoffmann's anodyne, ʒj (4.0), may be required in the bath. The average duration of the bath should be twenty minutes, longer if no reduction of high temperature. If the patient looks blue or shivers, he should be removed.

The Nauheim bath (Triton salts), advocated by William H. Thomson, especially if friction is combined, is superior to the Brand bath. In Fig. 210 is a portable tub, weight 5 pounds, excellent for private work.

Medicinal Treatment.—Antipyretics should be avoided. Though

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[1] In 100 cc. of milk is contained only 64 calories, or 640 calories per liter; 4 quarts (liters) of undiluted milk would not give more than 2800 calories required for a man weighing 154 pounds. The fallacy of pure milk diet is thus demonstrated. Rice, barley, and oatmeal average about *350 calories per 100*. If milk is given, its calorie value and digestibility are increased by these cereals.

Chantemesse has reported some results from serum-therapy, it is doubtful whether anything of value has yet been obtained, though interesting data have been reported on the injection of bacterial vaccines by Walters and Eaton.[1]

Bismuth subnitrate or subcarbonate, in a dose of gr. 15 to 20 (1.0–1.3), combined with the same quantity of saccharated pepsin, I employ every three hours, on an average of four doses a day. The bismuth helps control the ulcers. As urotropin causes disappearance of the bacilli from the urine, it would seem a logical remedy. Urotropin, gr. 5 to 10 (0.3–0.6), given preferably with equal quantities of sodium benzoate in Vichy which has become flat, is the best method. It should be administered three or four times a day. I have apparently seen some benefit in lessening the temperature and tympanites by this method. Acetozone (1 : 1000), given in divided doses, about 1 to 1½ quarts (liters) per day, has been favorably reported in some cases; each dose is flavored with orange juice.

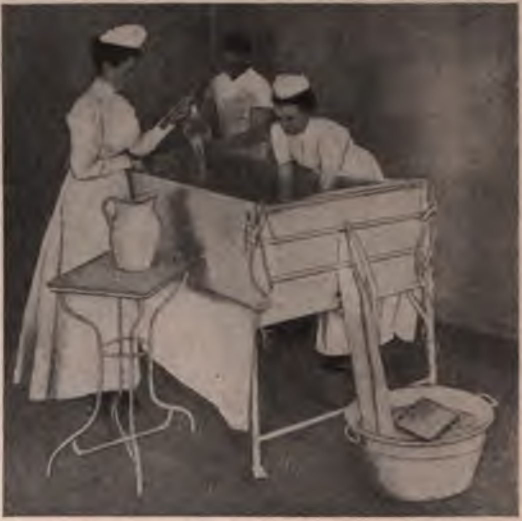

Fig. 210.—Chambers' portable bath-tub.

Tympanites.—Hot fomentations and turpentine stupes are employed. The ice-bag is best in many cases. A rectal tube may be inserted, or an enema containing oil of turpentine, ʒj (4.0), be given. Spirits of turpentine, ♏x to xv (0.592–1.184), can be given three or four times a day, or resin turpentine, gr. 3 (0.194), four times a day, or oil of cinnamon, ♏iij to v (0.178–0.296), every two hours. Charcoal, gr. 5 (0.3), bismuth subnitrate, gr. 15 (1.0), beta-naphtol, gr. 3 (0.194), or ichthalbin, ichthoform, or formidin, gr. 5 (0.3), every three to four hours may be substituted. Acute distention is relieved by enteroclysis. As acute dilatation of the stomach is often associated, lavage is also of value. This is especially true in distention with active hemorrhage.

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[1] Medical Record, January 16, 1909.

A thorough bowel action should at once be secured if there is no hemorrhage. Eserin, gr. 1/60 (0.00108) by hypodermic, may be of value.

As I order magnesium sulphate or citrate, ʒj (4.0), every second or third day in addition to the rectal irrigation, tympanites is rare.

In Fig. 211 is illustrated the correct position to relieve pressure from tympanites; pulse and respiration lessened 20 points as a result, and the tympanitic area in the thorax diminished 4 inches.

Diarrhea.—The bismuth preparations, chalk, and occasionally a little opium (see chapter on Diarrhea) may be required, and the enemata stopped for twenty-four hours, if the movements are excessive.

Constipation does not occur with the methods described.

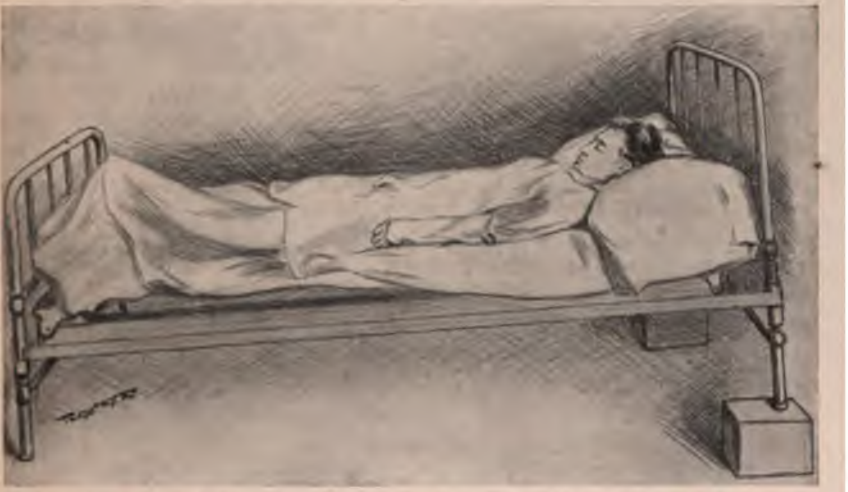

Fig. 211.—Postural treatment for acute dilatation of stomach and intestines in typhoid fever.

Hemorrhage.—Morphin, gr. 1/4 (0.016), by hypodermic, and the ice-bag immediately applied. Then lactate of calcium, gr. 15 (1.0), given with ℥ij to iv (60.0–125.0) of 5 to 10 per cent. gelatin solution; chlorid of calcium, gr. 10 (0.6), may be substituted. Thereafter lactate of calcium, gr. 10 (0.6), with ℥ij (60.0) of 10 per cent. gelatin every four hours. Ernutin, ♏v (0.296), may be given by hypodermic.

William H. Thomson recommends the following:

℞. Pulv. opii }
Argenti nitratis } .... āā gr. v (0.3)
Resin turpentine .... ʒij (8.0)
Liquor potassii .... ʒj (4.0)
Licorice pulv. .... q. s.—M.
Divide into 60 pills.
Sig.—Two pills every four hours. They may be given for a few doses at two-hour intervals.

Large doses of opium should be avoided, as they obscure symptoms.

Adrenalin (1 : 1000), ♏v to x (0.296–1.592), has been advocated by hypodermic, but it may increase pulse tension too markedly.

Hypodermoclysis, preferably in the iliolumbar region, as in Fig. 212, with normal salt solution, may be required, or even infusion.

At any time, on the appearance of blood in the stool or of suspected hemorrhage, stop enteroclysis and baths (if being given) *at once*.

Perforation and Peritonitis.—Early operation is indicated.

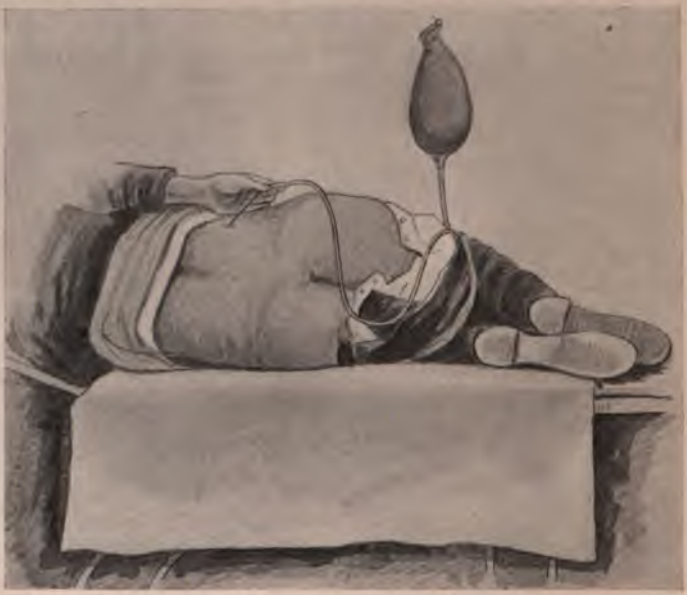

Fig. 212.—Hypodermoclysis in the iliolumbar region.

Heart Stimulants.—Strychnin sulphate, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0026), every three or four hours by hypodermic, or

℞. Pulv. camphor ....gr. viiss (0.5)
Sterile almond oil ....♏xx (1.184).
Sig.—One dose every four to six hours by hypodermic may be required, especially if there are complications.

Aromatic spirits of ammonia or Hoffmann's anodyne, in ʒj (4.0) doses, are useful in emergency

Caffein citrate, gr. 1 to 2 (0.065–0.021) every three or four hours, is of service.

Some advise alcohol, ℥viij to xij (250–375 cc.), in divided doses, but, like Seibert, I have not found it necessary. Large doses of tincture of digitalis may be added in the case of alcoholics. Careful stimulation with drugs, I believe, causes less strain on the kidneys.

The former views as to the value of alcohol as a food, or its power to increase the capacity for work, seem, by scientific research, to be pretty thoroughly exploded. Its use in the arctic regions, where food of high calorie value is at a premium, has been found to be deleterious. In sudden emergency, as a heart stimulant, it is of value, but its prolonged use as a circulatory tonic has been shown to be harmful. Proper individual dosage is difficult to estimate, and the strain on the already damaged eliminating organs in typhoid fever is severe. F. Lee has, moreover, demonstrated that the use of alcohol causes muscle fatigue.

For Nervous Symptoms.—Warm or cool packs with ice-bag to the head, and at times bromids or opiates.

For Sleeplessness.—Sulphonal, trional, or veronal, gr. 10 (0.6).

Bacilluria.[1]—Urotropin and benzoate of soda, of each gr. 10 (0.6) t. i. d.

Care should be taken to guard against bed-sores. Tender toes should be protected from the weight of the sheets, and hyperextension of the feet should be avoided. In some cases a water- or air-bed or an old sheepskin spread under the patient, as suggested by Thomson, may be required.

For Renal Insufficiency.—Cream of tartar lemonade: Cream of tartar, ʒj (4.0); juice of 2 lemons; saccharin, gr. j (0.063); water, 1 quart (liter). Drink in divided doses during the day. Recurrent enteroclysis with normal saline solution at 120° F. and proctoclysis are useful. Caffein citrate, gr. v (0.5) t. i. d., is of value. Hypodermoclysis may be necessary.

Colitis should receive treatment as described in that chapter. Delafield[2] has secured results in membranous colitis by irrigation with 2 quarts (liters) of bichlorid of mercury (1 : 10,000) with the recurrent tube.

Complications should receive appropriate treatment.

Convalescence.—It is usually preferable to have a normal temperature for a week before commencing with solid food. Soft-boiled eggs, milk toast, jellies, and a little scraped beef should be first tried.

In some *prolonged cases*, with temperatures of 99° to 100° F., cautious feeding may be attempted with the above materials. I have seen temperature fall as a result.

Bacilluria I believe a frequent cause of slight persistent temperature. This should receive treatment as already described. The patient should first sit up for a brief period about the tenth day of normal temperature.

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[1] In infection of the kidneys by the colon bacillus, Wm. H. Thomson advises active catharsis, followed by frequent doses of urotropin and sodium benzoate, āā gr. x (0.6), every three hours. If not retained by mouth, give by enema.

[2] Enteroclysis, Hypodermoclysis, and Infusion (Kemp).

CHAPTER XXVI

INTESTINAL HEMORRHAGE; INTESTINAL ULCERS; DISEASES OF THE BLOOD-VESSELS (EMBOLISM AND THROMBOSIS)

INTESTINAL HEMORRHAGE

It would seem advisable to refer to the various causes of intestinal hemorrhage for the purpose of diagnosis. Among such are dysentery, typhoid, yellow fever, malarial poisoning, ulceration from various causes, cancer scurvy, purpura, traumatism, volvulus, intussusception after reduction of a strangulated hernia, excessive use of laxatives, hemorrhoids, injury (traumatism), venous hyperemia of the intestines due to diseases of heart or lungs, stasis or obstruction of the portal system, such as cirrhosis of the liver, injury from dried scybalæ, ankylostoma, isolated venous varicosities, arterial aneurysms of the intestinal wall, aneurysms in adjacent arteries, as of the hepatic, from which blood may enter the bile-passages and be passed by the bowel.

A peculiar type of enterorrhagia occurs with no anatomic changes, but there is probably an alteration in the walls of the blood-vessels. Thus, intestinal hemorrhages have occurred in phthisis when there were no ulcers.

Intestinal hemorrhages in pernicious anemia, leukemia, scurvy, morbus maculosus, septicemia, icterus, phosphorus-poisoning, intermittent fever, and erysipelas probably belong to this class. Amyloid degeneration may be complicated by intestinal hemorrhages. Some believe vicarious hemorrhage may take the place of menstruation.

There may be collapse, with all the symptoms of internal hemorrhage, and blood may or may not pass from the rectum. The blood may be bright red, brown, or like coffee-grounds.

No visible blood may appear in the stools and be detected only by tests for occult blood.

Microscopic examination may show the presence of red blood-corpuscles and hematin.

Treatment.—The immediate hemorrhage should be treated as described under Hemorrhage in Typhoid, and the cause should be determined and treated.

ULCERS OF THE INTESTINES

SIMPLE DUODENAL ULCER

(*Synonyms.*—Duodenal Ulcer; Ulcus Duodeni Pepticum—Leube; Round or Perforating Duodenal Ulcer.)

This is characterized by a defect of the mucous membrane of the duodenum, usually tending to run a protracted course, though in some instances it may be acute. It presents the appearance and characteristics of ulcer of the stomach.

Etiology.—This is practically the same as gastric ulcer, being caused by the action of the acid gastric juice upon the mucous membrane of the duodenum, whose nutrition and vitality have previously been impaired as a result of circulatory derangement. Chlorosis, however, seems to play no part in its production.

Duodenal ulcers occur from burns of the abdomen, but are not correctly classified under peptic duodenal ulcer. Duodenal ulcer is less frequent than gastric ulcer, but is much more frequent than is generally supposed. Undoubtedly many cases have been diagnosed as gastric ulcer when it was a duodenal ulcer, lying close to the pyloric ring. William J. Mayo has demonstrated that many pyloric ulcers are found to have their origin in the duodenum, and in many cases it is impossible to differentiate between the two conditions. Trier places it as 1 to 9 gastric in frequency; while Andral as 1 to 40 gastric; while Starke places it at 1 to 12 gastric.

Age.—It occurs more frequently between the ages of fifteen and sixty. The average age of death in 127 fatal cases was thirty-eight years (Rolleston), but the Fenwicks show that in the acute cases 68 per cent. prove fatal between fifteen to thirty years, and in chronic cases, between thirty and thirty-five.

Duodenal ulcer occurs quite frequently in young children of one to ten years, and has been found in the newborn infant.

Gastric ulcers occur less frequently in young children.

Sex.—The proportion of *males affected by duodenal ulcer is greater than the females.*

Weir reports 30 women in 176 cases; Collins, 52 women in 257 cases; others give the rates from 5 to 1 to 3 to 1.

Pathology.—It resembles the gastric ulcer, having a punched-out appearance; it is oblong or oval, having a funnel shape, and extends to a variable depth. It is only irregular in shape when several ulcers have coalesced. It varie in size from a pea up to a silver dollar. William Mayo classifies them surgically as indurated and non-indurated. In the former case they have a callous margin. The base is formed usually of the intestinal wall (muscle), or at times by adhesions to adjacent organs.

Site of the Ulcer.—It is usually present in the upper 2 inches of the duodenum, in the ascending part near the pylorus, or in the upper horizontal part; less frequently in the descending part, and rarely in the third horizontal part. If the ulcer is situated near the diverticulum of Vater, through cicatricial contraction it may occlude the orifices of the bile- and pancreatic ducts and produce complications.

Usually there is one ulcer, more rarely, two to four. They vary in size from a lentil to a dollar.

Complications.—Stenosis of the duodenum near the pylorus or at a greater distance may occur, with resulting dilatation of the stomach and symptoms of benign stricture, the same as in the stenosis of gastric ulcer. Jaundice, distention of the gall-bladder, and inflammatory conditions therein may result from stenosis blocking the common bile-duct. Atrophy of the pancreas may take place from closure of the pancreatic duct. Fatal hemorrhage may occasionally occur. The type of hemorrhage is usually severe.

In acute cases quite frequently perforation and general peritonitis occur, with death from shock or peritonitis. If the process is more slow, there may be circumscribed peritonitis with abscess. There may be adhesions with other organs and ulceration involving the liver, gall-bladder, pancreas, aorta, portal vein, or hepatic artery. Subphrenic abscess may result.

Thrombosis of the portal vein has also occurred through deep cicatrization of an ulcer. The blood-vessels in the upper and descending duodenum have frequently been eroded. Cancer, developed on the base of a duodenal ulcer, has been reported by Arnold, Eichhorst, and S. Fenwick.

Clinical Aspects.—Occasionally the cicatrix of an old ulcer may be found at autopsy, though there have been no symptoms during life. Other patients may be apparently in perfect health, when suddenly a severe and dangerous intestinal hemorrhage takes place, or perforation and peritonitis.

Symptoms.—In some cases there are quite marked symptoms. The patient may complain of tension and pressure in the epigastric region that may occur without apparent cause, or after eating, or on palpation. In many cases there are paroxysmal attacks of pain that may be quite violent, which are difficult to distinguish from cardialgia or gall-stone colic. They are more frequently to the right of the linea alba and extend to the right, in a line below the liver. As a rule, the pains do not radiate to the back as in gastric ulcer, but rather downward in the abdominal cavity. There are intervals free from pain. Pain usually occurs from two to three hours after meals. Some claim the period of pain is later, four to six hours after eating, which aids diagnosis. I have not found this to be so.

Though the region near the pylorus is found to be slightly sensitive on pressure, there does not seem to *be the usual circumscribed* and *very sensitive spot*, as found in gastric ulcer. In some cases there may be continuous pain and tenderness, probably due to local peritonitis.

In others there is a boring or gnawing sensation in this region. Vomiting, which relieves the pain of gastric ulcer, is said not to do so in duodenal ulcer. Fluid given at the time of pain is said to lessen it by causing reflex closure of the pylorus or by diluting the excess of acid. The appetite and stools may be normal. Constipation frequently occurs, diarrhea rarely. The general condition of the patient is often good and there may be no great loss of weight.

Vomiting is rare, unless there is stenosis with gastric dilatation.

Gastric Contents and Vomitus.—In my own experience I have generally found hyperchlorhydria present, which corresponds with Boas' findings. This is in the uncomplicated cases. Normal contents, hypochlorhydria, or even the absence of free hydrochloric acid and the presence of lactic acid have been reported.

Hemorrhage is a very frequent symptom and occurs in at least one-third of the cases. The number is undoubtedly much larger, if care were taken to examine the stools and gastric contents for occult blood[1] by Weber's or the benzidin test. The blood is most frequently passed by the bowel and less frequently vomited. There may be severe hemorrhage retained in the bowel, with all the symptoms of collapse, or blood passed of a tar-like or coffee-ground appearance.

The death-rate from hemorrhage is quite large, estimated at 13 to 33 per cent. in acute and chronic cases.

Jaundice[2] is rare. With perforation there are pain, distention, muscular rigidity, etc.

William Mayo has shown that the onset of perforation is extremely acute, and the early pain is just to the right of the median line, while in gastric perforation it is usually to the left of this line.

In duodenal perforation the contents gravitate at once to the appendical region, and simulate perforated appendix.

The **course** may be very acute or very chronic. Hemorrhage and complications are common.

Diagnosis.—In many cases differential diagnosis between *duodenal* and *gastric ulcer* cannot be made. Acute cases following burns are easy of diagnosis.

The following are of service: Duodenal ulcer is much more frequent in males; pain is more frequent to the right of the median line and does not radiate to the back; local circumscribed tenderness on pressure not as marked as in ulcer; vomiting rare, unless stenosis; melena quite frequent; hematemesis is rarer than in ulcer; patients generally healthy in appearance; intestinal hemorrhage very frequent; perforation very frequent; pain in some cases at a later period than with stomach ulcer; latent type with sudden hemorrhage more common than with gastric ulcer. Though Leube states that hyperchlorhydria is present in gastric ulcer and normal acidity or diminished acidity in duodenal ulcer, so far in the uncomplicated cases I have found hyperacidity. Occult blood[1] in the stool is quite diagnostic.

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[1] Occult blood at least can at some time be found in every case.

[2] The development of icterus, in a case presenting some of the symptoms of gastric ulcer, probably shows duodenal ulcer, if gall-stones can be excluded.

The **prognosis** is quite serious, especially in the cases with frequent recurrent hemorrhage. Relapses may occur in the apparently cured cases, but I have seen complete recovery follow proper treatment.

Treatment.—This is similar to ulcer of the stomach. For acute hemorrhage, ice-bag to epigastrium, morphin, gr. ¼ (0.016).

Trémoliére's solution—calcium chlorid (2 per cent. solution) in gelatin (5 per cent. solution)—℥j (30.0), every two to three hours.

Gelatin (10 per cent.) by mouth, ℥j (30.0), or 2 per cent. subcutaneously; calcium chlorid or calcium lactate, gr. 10 (0.6), by mouth or enema; or strontium or magnesium lactate by hypodermoclysis, gr. 15 to 30 (1.0–2.0), in ℥iv (125 c.c.) sterile water, or ernutin, ♏v (0.296).

Adrenalin (1 : 1000), ♏v (0.296) or more, has been recommended by mouth or hypodermic. It may too markedly raise pulse tension.

For collapse, rectal saline enema, proctoclysis, or hypodermoclysis, with camphorated oil or strychnin by hypodermic. Rectal feeding for twelve hours and white of egg and gelatin (cold) by mouth.

Subsequently, large doses of bismuth, and nitrate of silver should be given, as in gastric ulcer. Treat hyperchlorhydria, if present, and feed by Lenhartz's method, as for ulcer of stomach. Iron and arsenic for anemia. Regulate the bowels

Surgery.—Perforation requires immediate operation; for frequently repeated large hemorrhages and for gastric dilatation gastroenterostomy is indicated. Local abscess, adhesions, or obstructive jaundice require operation.

INTESTINAL ULCERS FROM CUTANEOUS BURNS

Ulcers from extensive burns (cutaneous) generally occur in the upper transverse duodenum, seldom lower down. Rarely an ulcer may occur in the stomach or other part of the intestines. There may be a single ulcer or five or six of them. There is considerable loss of tissue in some cases, in others erosions, and at times inflammation of the mucous membrane. The shape of the ulcer is irregular and dentate or long and narrow.

The course is very acute, the result generally fatal; hemorrhage or perforation occurring within one to two weeks after the burn or even within two to three days. The condition is probably due to septic embolism. Operate if there is perforation. In *mild cases* treatment is that of duodenal ulcer. Intestinal hemorrhage and local tenderness are the salient symptoms.

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[1] The determination of *occult blood in the stool*, and at times occasionally in the vomitus, is of valuable diagnostic import. In an obscure case at the Red Cross Hospital, a positive diagnosis, ulcer of the duodenum, was made chiefly on this point and confirmed at operation by H. Haubold. It served to exclude gallbladder disease and a gastric neurosis.

TUBERCULAR ULCERS AND INTESTINAL TUBERCULOSIS

Tuberculosis is a quite frequent cause of ulceration of the intestines. The infection may be: (1) Primary in the intestinal mucous membrane; (2) most commonly secondary to diseases of the lungs; and, rarely, (3) secondary to tubercular peritonitis.

Primary intestinal tuberculosis occurs most frequently in children, and with it may be associated tuberculosis of the mesenteric glands or tubercular peritonitis.

R. Koch believes that bovine tuberculosis differs from human tuberculosis, and that infection from diseased milk or milk of tuberculous cattle hardly ever occurs, and it is unnecessary to take any precautions. Von Behring takes the opposite view.

In the Charité Hospital in Berlin there were only 10 cases in ten years. In 3104 cases of tuberculosis in children, there were 16 of primary infection. There have been investigations recently reported which are suggestive that infection through the intestines is more common than we suppose, notably, Macfayden found tubercle bacilli in the mesenteric glands of 5 out of 20 children post mortem, with no tubercular lesions elsewhere; and Ravenal in 8 cases out of 25.

Recent consensus of opinion holds that primary intestinal infection occurs through tuberculous milk. Milk from an infected nurse or mother may also be a cause. The meat of tuberculous cattle probably plays a lesser rôle, as it is generally thoroughly cooked. There is no reason why other raw food products may not be occasionally a source of infection, especially if exposed to infection by flies or sources of contamination. In adults primary intestinal tuberculosis is rare (in the Munich Pathologic Institute 1 in 1000 cases). The lower ileum is usually first involved and then the rest of the small intestine and colon.

Symptoms.—The condition may begin with irregular diarrhea, slight fever, and colicky pains. Rarely, hemorrhage may be the first symptom. At first the symptoms may simulate a chronic catarrh. Until subsequent emaciation becomes marked or an involvement of the lungs occurs, the condition may not be suspected. The stools in every doubtful case should be examined for Koch's tubercle bacillus, which is diagnostic. The tuberculin test, by injection or ocular, should be made.

A deceptive condition is when the ulceration begins in the cecum, and there are symptoms suggestive of appendicitis, with tenderness in the right iliac fossa, constipation, or irregular diarrhea. Osler reports in his primary cases of intestinal tuberculosis occasional fatal hemorrhage or perforation, with the formation of pericecal abscess or perforative peritonitis, or, rarely, partial healing, with great thickening of the intestinal walls and narrowing of the canal (chronic hyperplastic tuberculosis).

Tubercle bacilli in the stools are diagnostic of these conditions when found; otherwise they are difficult of diagnosis unless by operation. The symptoms and treatment of tuberculous ulcer (tuberculosis) of the intestines will be described at the end of this chapter.

CATARRHAL AND FOLLICULAR ULCERS

These types of ulcer occur in the course of catarrhal inflammation of the intestinal mucous membrane, and are described in that chapter.

ULCERATIVE COLITIS

This special form of ulceration of the colon was first described by Hale White, colitis occurring especially in institutions and insane asylums. Probably the greater percentage of these so-called cases of colitis, such as occur in institutions, are true dysentery.

Vedder and Duval,[1] while working under Flexner, found that institutional outbreaks were due to Bacillus dysenteriæ. Osler and J. P. Tuttle differ in this regard and classify it as non-dysenteric. The symptoms and treatment are of dysentery.

STERCORAL OR DECUBITAL ULCERS

These ulcers are produced by the pressure of hardened and stagnating feces on the mucous membrane, resulting in necrosis and purulent inflammation. They develop particularly in the cecum, flexure of the colon (hepatic and splenic), sigmoid flexure, and the rectum, where stasis is most apt to occur. Fecal concretion in the appendix is fairly common and may produce ulcer and appendicitis. They sometimes develop in chronic intestinal stenosis above the seat of stricture.

ULCERS IN ACUTE INFECTIOUS DISEASES

Under this group are the specific ulcers of typhoid and dysentery; diphtheritic ulceration; ulcers of sepsis; rarely duodenal ulceration in erysipelas; in varioloid; ulcer of the duodenum in pneumonia, a rare occurrence. In acute pemphigus and in pellagra isolated cases have been reported.

ULCERS IN CONSTITUTIONAL DISEASES

In acute leukemia, lymphatic tumors may occur in the intestines, especially in the ileum, and break down, with the production of ulcers. This is rare in the chronic type. Intestinal ulceration has occurred in scurvy and a few isolated cases are reported in gout.

TOXIC ULCERS

Intestinal ulcers occur in nephritis, with uremic symptoms associated with the intestinal catarrh. They lie chiefly in the large intestine.

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[1] Journal Exp. Med., Feb. 5, 1902, vol. vi.

In poisoning with mercury they are also found, even when it is not administered by mouth or rectum, but by inunction.

Changes in the blood have been held responsible for these conditions. With nephritis the intestinal catarrh is probably a factor; it is believed by many that the mercury reaches the intestines by excretion in the bile, and produces direct inflammation with resultant ulcer, in which decomposition or intestinal bacteria play a part, since we find in some cases a pseudodiphtheritic membrane.

SYPHILITIC, GONORRHEAL, AND CANCEROUS ULCERS

Syphilitic ulceration of the intestines is rather rare. In the small intestine it may be found in a young infant with inherited syphilis. Ulcers originate in the lymphatics or in the mucosa or submucosa, from a gumma which gradually breaks down. A few cases of syphilitic ulcer of the small intestine in an adult have been reported by Klebs, Osler, and Birch-Hirschfeld. These ulcers are rarely encountered in the large intestine, except in the rectum in its lowest part. Primary chancre of the rectum has been observed. If feasible, the test for the Wassermann reaction should be made to confirm the diagnosis. The physical examination and history of the case are important.

Condylomata and gummata may break down, and by cicatrization give rise to stricture of the rectum.

Virchow notes that syphilitic ulcers are of slight depth and have a smooth base. They are more frequent among women than men.

Polchen[1] found 190 cases of stricture among women out of 219 cases, but believes that many of these result from ulcerations caused by gonorrheal infection, from abscess of the Bartholin glands, or from fecal pressure (decubital ulcer), or local traumatism from the syringe-tip.

Ulcers from gonorrhea, traumatism, and hemorrhoids occur in the rectum and are referred to under Proctitis. Ulceration of intestinal carcinoma may occur, or metastatic nodules may break down and ulcerate.

INTESTINAL MYIASIS

A case of fatal ulceration of the colon due to maggots has been reported.[2]

GENERAL SYMPTOMS OF INTESTINAL ULCERATION

Our readers must take into consideration the special types of ulcer, their etiology, and the history of the case in making the diagnosis.

In some cases this is quite difficult, as the symptoms are not constant. The salient diagnostic points of intestinal ulcer are as follows:

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[1] Virchow's Archiv., Bd. 127.

[2] Schlesinger, Wiener klin. Wochenschr., January 9, 1901.

1. The appearance of pus in the stool. This is frequently in small quantity and in the form of minute grayish-white lumps, which appear under the microscope as closely aggregated masses of pus-cells, and are especially important. At times it is only microscopic.

2. Necrotic pieces of the intestinal mucosa, such as shreds of tissue from the intestinal wall, which must be differentiated from membranous or shred-like particles of food, occur in some cases.

3. The appearance more or less frequently of blood in the stool, unchanged and in large amount as pure blood, or changed, coffee-ground, or *occult blood.* In some cases blood is only shown under the microscope, or by hematin crystals, or by Weber's or the benzidin test. Gastric ulcer and vicarious hemorrhage must be excluded.

4. The constant appearance of tubercle bacilli in the stools in connection with diarrhea and increasing emaciation are diagnostic of tuberculous ulceration; also the tuberculin reaction.

5. Persistent diarrhea (with local tenderness) and pain of a greater or lesser degree over a definite region of the abdomen, extending over a considerable period of time, associated with the presence of pus and blood in the dejecta.

6. Tenesmus, with pus and blood in the stool, suggests ulcer, probably in the rectum, and visual examination makes the diagnosis.

The presence of mucus is not diagnostic of ulcer.

Diarrhea.—This is present in a large number of cases. It is dependent on the site of the lesions; ulcerations of the small intestine, cecum, or ascending colon probably do not produce diarrhea unless there is a complicating catarrh, amyloid degeneration, or some special infection like typhoid fever. Even in the latter we have constipated cases. Ulcers of the lower colon and rectum usually produce diarrhea, but even here it may occasionally be absent.

Blood.—Pure blood may be passed in large amounts in simple duodenal ulcer from burns and in typhoid. Large hemorrhages at times occur from dysenteric ulcers. The hemorrhages from catarrhal and tuberculous ulcers are, as a rule, not as large.

The blood may be bright red or dark, or of coffee-ground color, or may be only determined microscopically as blood-corpuscles or hematin crystals, or by tests for occult blood.

Intestinal ulcer may be present without hemorrhage, and other conditions may produce intestinal hemorrhage,[1] such as liver cirrhosis, etc.

Pus is diagnostic of intestinal ulcer. It is also found in connection with ulceration accompanying neoplasms of the intestines, and in abscesses which open into the intestines. It is generally in small amounts. Occasionally no pus is found, as in the case of duodenal ulcer; or the ulcer may be single and high up, so that the pus will disappear. If small grayish-white specks are discovered in the stool and under microscopic examination they are found to be pus, the diagnosis of ulcer is established. Pus is often only found by the microscope.

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[1] Repeated examinations, however, will generally show occult blood.

Mucus found mixed with the intestinal contents is the result of *associated intestinal catarrh.* We can draw some conclusion from the relative amount of pus, blood, and mucus. *Mucus is never diagnostic of ulcer.*

Pure pus is also found in a diphtheritic process of the bowel or from perforating abscess.

Blood, pus, and mucus occur in dysentery and in carcinoma of the lower colon or rectum.

Shreds of Tissue.—They consist of mucous membrane and are differentiated from particles derived from food; they occur most frequently in dysenteric ulcer and not in the slower type, as in tuberculosis; or in the more rapid, as in typhoid.

Tubercle Bacilli.—Generally diagnostic of tuberculous ulcer, in connection with the other symptoms. Rarely sputum may be swallowed and pass through without infection. Absence of bacilli does not always prove absence of tuberculous process. The injection of tuberculin or the ocular test (conjunctival) aid diagnosis in the doubtful cases.

Pain.—This is occasionally absent. If pain exists in a circumscribed spot for a long time and is increased on pressure, it is probably due to ulcer. It may at times be caused by local peritonitis.

Ulcers of the rectum produce tenesmus, which is quite characteristic. Rectal examination should always be made.

Fever is present in the tuberculous type, dysentery, etc., but not in all types of ulcer, being dependent on the etiologic cause.

General Nutrition.—This may not be disturbed by a few small ulcers, but marked ulceration, especially of the tuberculous type, leads to great emaciation, as the intestinal contents are rapidly propelled and also normal absorption is interfered with.

Perforation with general peritonitis, local peritonitis, or encapsulated abscess may occur. Stricture may result from ulcers.

Prognosis.—The prognosis depends on the etiology.

TREATMENT

Hemorrhage should be treated as in that from duodenal ulcer. The primary cause should receive appropriate treatment; in uremic ulcers, the nephritis; in syphilitic ulcers, by hypodermics of bichlorid of mercury; mercurial inunction; protiodid, bichlorid, and iodid of potassium.

Heat or cold can be applied for the pain and rest in bed for the severe cases.

In tuberculous ulceration, out-of-door life and change of climate are important. Beechwood creosote, ♏ij (0.118) t. i. d., guaiacol carbonate, carbonate of creosote, and creosal (tannosal) are useful. Average doses of these remedies are gr. 10 (0.6) t. i. d.

Diet is important; it should be non-irritating and chiefly liquid, such as milk, kumyss, matzoon, bacillac, lactone-buttermilk, raw eggs beaten in milk; soft-boiled eggs, broths, chicken soup, mushes, etc. Fats, such as butter, emulsion of mixed fats (Russell's), cream, etc., are of value.

Some cases can take sweetbread, scraped beef, calves' brains, cocoa, tea or weak coffee, milk-toast, etc.

Sanatogen (flavored) and malt-tropon can be given in broths or soups.

Compound tincture of catechu, chalk mixture, opium, etc., are useful for severe diarrhea. The following are excellent:

℞. Comp. tinct. opii }
Bismuth. subnit. } .... āā ℥ss (16.0)
Mist. cretæ .... ℥ij (60.0)
Aq. destil. .... q. s. ℥iv (125.0).—M.
Sig.—Shake. Dose, ʒij (8.0) every three hours.

℞. Tr. opii .... ♏x (0.59)
Mist. cretæ .... ʒj (4.0)
Comp. tinct. catechu .... q. s. ℥ij (8.0).—M.
Sig.—Dose, ʒij (8.0). Administer every three hours.

Bismuth subnitrate, gr. 15 to 30 (1.0–2.0), three or four times a day, or bismuth subcarbonate or bismuth salicylate, gr. 10 to 15 (0.6–1.0), may be substituted. Avoid opiates as much as possible.

Tannigen, bismuth subgallate, tannalbin, and tannocol are useful in average doses of gr. 10 to 15 (0.6–1.0) three or four times a day.

High injections of silver nitrate, 1 : 3000; thymol, 1 : 2000; salicylic acid, 1 : 300; boric acid, 1 : 500; tannic acid, 1 : 1000; and protargol, 1 : 1000, are of service in rectal and colonic ulcers.

Carbolic acid should not be used, and bichlorid of mercury (1 : 10,000) by recurrent irrigation only in typhoid in the diphtheritic form of colitis. Special local treatment is necessary for proctitis, as already described.

DISEASES OF THE BLOOD-VESSELS; EMBOLISM AND THROMBOSIS OF THE MESENTERIC ARTERIES AND VEINS (INFARCTION OF THE BOWEL)

Embolism and thrombosis of the mesenteric arteries are comparatively rare affections.

When the mesenteric vessels are blocked by emboli or thrombi, infarction follows in the territory supplied by the vessel, which may continue to gangrene or perforation and peritonitis. If only a few small vessels are occluded there may be few if any symptoms, and the circulation may be re-established. Welch[1] states that about 70 cases of embolism or thrombosis of the mesenteric arteries have been published, while Gallavardin[2] has collected 83 cases, of which 63 were of embolism.

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[1] Allbutt's System of Medicine, vol. ii, 1894; also vol. iv.

[2] Gaz. des Hôp., Paris, 1901, p. 929.

As the clinical symptoms of obstruction of the mesenteric arteries and veins can scarcely be differentiated during life, the symptoms being much the same, and since careful study of their etiology may aid in diagnosis, it is advisable to describe them together. Including both arterial and venous mesenteric obstruction, Jackson, Porter, and Quimby have studied about 30 cases in Boston, and have collected 214 cases in all.

Etiology.—*Embolism and Thrombosis of the Mesenteric Arteries.*—In a majority of cases occlusion is due to embolism and rarely to thrombosis.

The chief source of the emboli are traceable to endocarditis (valvular lesions) or atheroma of the aorta, or rarely from a pulmonary thrombosis or aneurism of the aorta.

Thrombosis.—Local changes in the vessels, as endarteritis (described by Litten), syphilitic endarteritis, injury and pressure from calcareous glands, have been given as causes. Rolleston refers to peri-arteritis nodosa, a rare condition in man, with the production of multiple aneurysms, which produces pain, colic and diarrhea, with ulceration. The superior mesenteric artery is more often affected. Verminous aneurysms cause infarctions in the horse.

Etiology of Thrombosis of the Mesenteric Veins or the Portal Vein.—Welch[1] has collected 32 cases and has demonstrated that the superior mesenteric vein is more often affected than the inferior mesenteric. Among the causes are pressure on the portal vein, as in cirrhosis or cancer of the liver; neoplasms of the abdomen; chronic peritonitis, with formation of constricting tissue; local pressure r incarceration of the intestines; suppurative inflammation of the portal system as a result of infection, as in appendicitis, intestinal ulcer, or dysentery. Traumatism may produce inflammation and thrombosis. Mayland[2] shows that it may occur after operation; from tuberculous peritonitis, or diseased mesenteric glands. Hemorrhagic infarction of the intestines, as a rule, occurs with venous thrombosis, and the general symptoms are the same as due to arterial occlusion; hence, the comparative study of the etiology of these different conditions is important for diagnosis.

We would note that in a few rare instances Welch and Rolleston have reported exceptional conditions; the former, that acute portal obstruction has caused hemorrhagic infarction of the intestines, without thrombosis of the mesenteric vein; and the latter, thrombosis of both veins without infarction.[3]

Pathology of Occlusion of the Superior Mesenteric Artery.—The changes resulting from occlusion of this vessel or its branches are hemorrhagic infarcts and peritonitis. If only small branches are occluded the results are embolic or thrombotic *ulcers of the intestines*, to which article I refer my readers.

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[1] Allbutt's System of Medicine, vol. v.

[2] British Medical Journal, 1901, vol. ii.

[3] Rolleston, Trans. Path. Soc., vol. xlii.

The transverse duodenum, ileum, jejunum, caput coli, ascending and transverse colon are supplied by this vessel, and lesions of the intestines occur in that portion of the bowel supplied by the occluded branch or branches. It usually involves a continuous segment of the gut, quite frequently in the lower jejunum and ileum; but if smaller vessels or branches are occluded, there may be lesions scattered in several places over the bowel, with healthy segments between them.

The superior mesenteric artery has such long and small branches that it acts functionally, like a terminal artery, so sufficient blood cannot be supplied to the intestines if it or one of its branches be occluded.

Welch and Mall[1] have demonstrated experimentally that if a branch be occluded, the blood which produces the hemorrhagic infarction is derived from anastomosing arteries and not from regurgitation from the portal vessels, as was formerly supposed.

The following are the clinical appearances in the intestines: Arteries empty, except at obstruction. There are venous hyperemia; edema and necrosis; hemorrhages in the mucous membrane and mesentery. The mucosa is a dark red and finally becomes necrotic, of a brownish-green appearance. The intestinal canal contains extravasated blood, either fresh or tarry looking. Necrosis of the intestines is present.

The serous coat is inflamed, not only in the affected area but also over the healthy intestines, and the coils may become adherent and be covered with fibrin. There may be a blood-stained or purulent exudate in the peritoneal cavity. Occasionally gas may be present in the cavity due to the Bacillus aërogenes capsulatus or emphysema of the mucosa.

Clinical Symptoms.—Kussmaul and Gerhardt[2] first clearly described these some years ago.

There are two types, the most frequent characterized by *hemorrhage from the intestines;* the second, *simulating intestinal obstruction,* with or without peritonitis.

Usually the onset is sudden, frequently with violent, colicky pains in the region of the umbilicus, which may gradually become diffuse, and at the same time there is tenderness on pressure over the abdomen. We must remember that in some cases there is an absence of pain.

Vomiting may accompany the pain. Diarrhea may begin shortly after the pain and become extreme—mucous, watery, and at last bloody—or an intestinal hemorrhage may occur at the commencement, with dark-brown or tarry stools, which at times may have a fetid odor. The blood may not always be voided, but retained, and the patient may have the symptoms of hemorrhage—collapse, cold extremities, rapid and feeble heart, and subnormal temperature.

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[1] Johns Hopkins Hosp. Reports, vol. i.

[2] Würzburger med. Zeitschr., 1863 and 1864, Bd. iv and v.

In the other type of cases the patient may have signs of acute intestinal obstruction; a history of constipation for some days; distended and painful abdomen; vomiting, at times feculent; severe abdominal pains; collapse and peritonitis. The attack is clearly due to intestinal paralysis.

Diagnosis.—The following may be of service: An intestinal hemorrhage from no apparent cause; colicky pains of great severity; tympanites and tenderness; evidences of effusion into the peritoneal cavity; the discovery of simultaneous embolism in other vessels or of an endocarditis. In many cases, however, the salient symptoms are not all present; and in the type simulating obstruction the true cause cannot be determined.

Prognosis.—This is generally fatal in occlusion of the superior mesenteric artery. In rare instances recovery may be possible.

Treatment will be described at the end of the chapter.

Embolism and Thrombosis of the Inferior Mesenteric Artery.—In 83 cases of Gerhardt, in only 5 instances was the inferior mesenteric artery obstructed; in 4 cases by emboli and in 1 by thrombosis; and in 2 of these the superior mesenteric artery was also obstructed. *This condition is rare.*

Symptoms.—The chief symptoms are pain, tenesmus, and bloody stools. Gerhardt states that the blood is bright red and that in obstruction of the superior vessel it is darker, but I believe it generally dark. The descending colon, sigmoid, and rectum, or parts of these are affected, the mucosa becoming red, succulent, and containing effusions of blood. The mucous membrane may be loose and detached in places and hemorrhagic. There are not the serious lesions, as a rule, as in occlusion of the superior mesenteric artery. The artery (inferior mesenteric) is not an end artery functionally, and the circulation is generally re-established. Rarely there may be infarction or gangrene with perforation.

Thrombosis of the Mesenteric Veins or Portal Vein.—This condition is extremely serious.

Anatomic Findings.—The arteries are distended (thus differing from the superior mesenteric artery obstruction); thrombotic processes are found in the mesenteric vein or branches or in the portal vein. The intestines show similar conditions to obstruction of the arteries—infarction, necrosis, hemorrhage, peritonitis, etc.

Symptoms.—The clinical picture may present the same type as in obstruction of the mesenteric arteries—colicky pains, tenderness, blood, diarrheal movements, etc. Rarely there may be hematemesis and occasionally constipation. The etiology, such as appendicitis, diseases of the liver causing pressure on the portal vein, abdominal neoplasms or constricting bands, producing pressure, etc., and the exclusion of the causes of embolism, may aid our diagnosis.

Treatment.—Temporarily, ice-bag to abdomen; stimulants, morphin, gr. ¼ (0.016), and ernutin, ♏v (0.296), by hypodermic; gelatin solution, calcium chlorid, or lactate of calcium to check hemorrhage, as described in Dysentery.

Immediate resort to laparotomy and resection of the diseased area is indicated, if possible. Cases of recovery after this procedure have been reported. Elliott resected 48 inches of intestines in one case with recovery.

CHAPTER XXVII

NEOPLASMS OF THE INTESTINES

MALIGNANT GROWTHS

(*Synonym.*—Neoplasmata Maligna Intestini.)

THOUGH many have claimed that intestinal cancer is a comparatively rare disease, recent statistics show that it has increased in frequency and is more common than has been generally supposed.

Among the malignant tumors met with in the intestines, carcinoma is by far the commonest. Sarcoma and lymphosarcoma are also found, and will be described in a separate section.

CARCINOMA OF THE INTESTINES

Etiology.—The cause of intestinal cancer, like that of cancer of other organs, is still unknown. There have been many theories advanced, such as the embryonal origin, parasitic infection, the theory of irritation, etc. The traumatic theory (irritation as the cause), in so far that it might *hasten the development* of the condition, is afforded a certain amount of plausible support, in view of the fact that the disease occurs most frequently in those parts of the bowels in which the feces are retarded, and hence act as a source of irritation. The points of selection for the development of cancer are the same as those in which the so-called decubital or stercoral ulcers most frequently develop, and we may assume they probably originate from the cicatricial tissue of these ulcers, in some cases at least, or on the base of other ulcers.

Billroth has found a carcinoma in the scar of a dysenteric stricture, just as it occurs in the case of carcinoma engrafted on the scar of a stomach ulcer. It will probably eventually be shown to be due to some specific organism in my opinion.

Sex.—Carcinoma of the intestines appears to be somewhat more common in men than in women.

Age.—In general this lesion is most common between the fortieth and sixty-fifth year.

Contrary to cancer developed in other regions, cancer of the intestines has been encountered quite frequently before the fortieth year. It has been found even in children; several cases being reported at the ages of eleven to seventeen, and 2 cases in children of three years of age.

Maydl, of Vienna, has calculated from records that one-sixth of all cases of intestinal carcinoma occur between thirty and forty years, and one-seventh before the thirtieth year. It is important to remember that early occurrence is *fairly frequent.*

Situation of the Carcinoma.—Cancer of the bowel is rare in the small intestine, occurs quite frequently in the colon, and most commonly in the rectum.

From 1882–93, from autopsies in the Vienna General Hospital, Riegel states 5 cases were in the duodenum; 6 in the ileum; none in the jejunum; 1 in the vermiform appendix; 14 in the cecum; 63 in the colon; 40 in the sigmoid, and 114 rectal cancers.

At the same hospital, during twelve years, out of 254 cases of cancer of the intestines, Maydl found in *the living* that 224 were cancer of the rectum.

Bryant, in 110 autopsies, found carcinoma of the small intestine 6 times; 7 times in the cecum and ileocecal region; 19 times in the transverse colon, including its flexures; 78 times in the sigmoid flexure and rectum.

Leichtenstern's data show that 80 per cent. of all intestinal cancers occur in the rectum.

Other statistics are given. They all show the preponderance of rectal cancer.

Intestinal cancers are *almost always primary*, and secondary growth by metastasis is extremely rare.

It may occur through direct extension by continuity, as a cancer of the pancreas may extend to the intestines.

Primary carcinomata of the intestines often give rise to metastases in other organs. These are most frequently found in the lymph-glands, especially in the neighborhood of the neoplasm.

Secondary metastases in the liver are quite frequent, no matter where the situation of the carcinoma. The peritoneum, omentum, mesentery, and lungs may be involved, and occasionally the kidneys.

Hauser calls attention to certain peculiarities regarding the metastases in different forms of carcinoma of the large intestine. Colloid carcinoma chiefly involves the serous coat and metastases of internal organs are rare. Medullary tumors involve adjacent lymph-glands, while simple and scirrhous carcinomata, even when small, frequently cause *carcinoma of the liver.*

Morbid Anatomy.—Several varieties of carcinoma are found in the intestines, most often the cylindric epithelial-celled carcinoma (adenocarcinoma), starting in the cylindric epithelium of the intestinal glands (follicles of Lieberkühn). It occurs most frequently in the large intestine, as do medullary carcinoma and colloid cancer. More rarely the scirrhous carcinoma is found. The epitheliomatous chancroid occurs in the lower rectum at the anus, and it may involve the perineum and vagina.

In the small intestine the primary proliferation starts from the glands of Brunner; in cases developing from cicatrices, proliferation may start from glandular tubules which have grown deep down into

Perforation may rarely occur before the formation of adhesions, with resulting general peritonitis, or there may be a circumscribed abscess formed within the adhesions. Marked displacement of the intestines may be caused by the formation of these adhesions.

A carcinomatous peritonitis may be produced by extension from the serous layer of the intestines, accompanied by hemorrhagic exudation. Perforation into other organs which have become agglutinated to the bowel can occur; thus a fistulous opening forms between the colon and the stomach or bladder, or vagina or uterus, or between the large and small intestines, or from the bowel through the abdominal wall.

The omentum and mesentery may become infiltrated with cancerous masses. A band may be formed by the stiffened omentum, which may cause a knuckling or a twisted bowel. The mesentery may kink and twist the intestines.

Symptoms.—The symptoms vary, depending on the position of the neoplasm, the rapidity of its growth, and the character of the cancer. There may be symptoms at first of habitual constipation or of hemorrhoids, or of stenosis of the intestines, or of peritonitis, or icterus, or of cachexia of uncertain origin. It seems preferable to first review the general and then the local symptoms, depending upon the position of the tumor, as occurring with—

1. Carcinoma of the duodenum.
2. Carcinoma of the small intestine.
3. Carcinoma of the colon (cecum to sigmoid flexure).
4. Carcinoma of the rectum.

General Symptoms.—There are symptoms common to all cases of carcinoma, of which anemia and cachexia are the most important. As a rule they occur together, though one may develop before the other. In many patients weakness, pallor, and emaciation are the first signs noticed, and arouse the suspicion of a serious disease. There may be only slight local symptoms, moderate constipation, and a sense of weight or discomfort in the abdomen, with loss of appetite, coated tongue, and slight dyspeptic disturbances. There is loss of weight and a peculiar cachectic appearance. On the other hand, the local symptoms may be the more pronounced, or the local and general symptoms may occur together.

Quite frequently, especially in those cases of carcinoma which run a rapid course, fever is present. I have seen a number of cases in which it is entirely absent. It probably depends upon an ulcerative condition in the growth and absorption of toxic products. The tumor may cause symptoms in adjacent organs by dragging upon them or constricting them. There may be radiating pains from compression of nerves and also disturbances of the circulation, such as edema of the lower extremities.

Symptoms of chronic intestinal obstruction are frequently present, but not in all cases. The symptoms may develop gradually, the passing of a rectal bougie or introduction of a speculum should be employed. In women a vaginal examination should also be made. *I strongly deprecate the insertion* of the entire hand into the rectum under anesthesia, as has been sometimes advised.

When the cancer is fully developed we may have peritonitis, either local or general, as a complication. If the peritonitis is of a cancerous nature, a hemorrhagic exudation and the presence of nodules under the abdominal wall will indicate this fact. Acute perforative peritonitis will be indicated by the usual symptoms of this condition. On the other hand, we may have circumscribed adhesions with the presence of local fecal abscess.

Perforation may occur into adjacent adherent organs which will present special symptoms.

Among the chief communications are:

1. Communication between the colon and stomach. There may be a valvular communication or it may be free. If the direction of the passage is from the stomach into the colon, symptoms of lientery develop—undigested food, such as rice, potatoes, meat, etc., appear rapidly in the stools. Diarrhea occurs shortly after the ingestion of food with evacuation of solid contents. If the valvular action is in the opposite direction, feculent vomiting takes place. If the fistulous opening is perfectly free, then lientery and fecal vomiting occur together or may alternate. This combination of symptoms is pathognomonic. Lavage, especially if the fluid be colored with methylene-blue or carmin, gr. 3 (0.194), will demonstrate that the liquid rapidly escapes from the stomach, and is expelled from the bowel without being decolorized or discolored.

If the rectum is inflated with air or CO_2 the stomach will be distended with gas, or after injection of 1 to 2 liters (quarts) of colored fluid into the bowel some can be secured from the stomach by aspiration.

2. Communication between the rectum and bladder. Fecal matter and gas escape into the bladder and are voided through the urethra. Severe septic cystitis results, or the urine may escape from the bladder and be discharged through the stools. A carmine solution injected into the rectum or bladder will demonstrate the condition, and will appear respectively in the urine or feces.

3. Communication between the rectum, uterus, and vagina are also met with, and give rise to the passage of fecal matter through these organs, and also to severe inflammation. Injection of weak carmin solution or methylene-blue into the rectum will demonstrate the communication.

4. Communication between the bowel and abdominal wall. There may be a feculent or fetid discharge, or even of particles of fecal matter, if the communication is with the lower small or large intestines. If the communication is high up, it may be chylous or biliary. This condition appears usually in the last stage of the

Carcinoma of the small intestine (jejunum and ileum) is rare. The symptoms vary, depending on the position of the growth; the higher up, the more marked are the gastric symptoms; the lower down, the more severe the intestinal symptoms.

There may be anorexia and vomiting, or fair appetite and good stomach digestion, but obstinate constipation.

In both conditions there are anemia and cachexia, pain, attacks of colic and constipation, which may alternate with a diarrhea. There may occasionally be hemorrhages from the bowel if the growth is low down. The clinical symptoms of *stenosis* of the intestines may be present. The tumor is at times accessible to palpation, and is, as a rule, extremely *movable*. It may be so much displaced downward that it is difficult to draw an accurate conclusion.

Local Symptoms of Carcinoma of the Large Intestine.—Cachexia and anemia are present. Pain is a frequent symptom; it may not be present at first, usually appears later in the disease, and becomes localized; in some cases, running an acute course, no pain appears until the symptoms of acute obstruction begin. Pain is usually localized; it may appear near or at the region of the growth, or occasionally directly opposite, in the abdomen.

The pain may not be severe, but may be more a sense of discomfort. There may be neuralgia of the sciatic or anterior crural nerves. Later pain usually becomes localized, and is increased on pressure, even if the tumor cannot be palpated. It may be due to local peritonitis near the tumor.

Attacks of colic often occur, local or diffuse. The pains may be quite severe, are accompanied with constipation, and are relieved by diarrhea or by the passage of flatus. These attacks of colic are frequently caused by the commencing obstruction, and show gradually progressive stenosis. Peristaltic and tetanic movements of the intestines are often associated. Complete obstruction may suddenly develop, or there may be a gradually progressive stenosis with its symptoms.

Constipation is one of the marked symptoms; in some cases it may be the first, and becomes gradually progressive.

Ten to twenty or even eighty-eight days have passed, according to various observers, before the bowels moved spontaneously or by artificial means. These were, of course, extreme cases. The majority of patients have symptoms of commencing or complete obstruction after coprostasis has lasted a week or ten days. This has been my experience in consultation practice. Recently I saw a case for the first time on the fourth day of coprostasis when acute symptoms were present. Loss of appetite, tension, fulness in the abdomen, and pain accompany the constipation. Spontaneous diarrhea may relieve the condition. If this does not occur, or relief is not afforded artificially, gradual occlusion will take place with its typic symptoms.

In some patients diarrheal movements may occur for several weeks due to the catarrhal condition of the bowel.

patients are generally over sixty years of age, fat and flabby, or they have been fat previous to emaciation. There is often a history of acute left-sided inflammation and generally of prolonged constipation. One must remember the possibility of this condition. *Blood and pus, as a rule, are absent from the stools in these patients.* Often it is impossible to differentiate these conditions, even after resection, except by the microscope.

Cachexia and anemia are always present in cancer.

Primary Carcinoma of the Appendix.—This is rare; most of the cases give symptoms of relapsing appendicitis or appendicitis; 53 per cent. are under thirty years, and 24 per cent. under twenty. This condition has only been detected after operation on the appendix. The carcinoma is spheroidal celled.

Carcinoma of the Rectum.—This is the most frequent type of cancer, is more readily diagnosed, and more amenable to treatment if early operation is performed. Many of the tumors are within reach of the finger. The symptoms resemble those of carcinoma of the colon, though there are certain signs peculiar to this condition. Rectal examination should always be made.

Pain is more pronounced in rectal carcinoma, both local and radiating to the sacrum, back, bladder, genitals, and to the sciatic nerve; there is often a desire to urinate. The pain is usually worse during defecation if the growth is low down, and it may even be agonizing in character, so much so that the patients try to retain the bowel contents as long as possible.

Marked tenesmus is present, and if ulceration, mucus, blood, and pus are evacuated. Constipation is usually present. The higher up in the rectum, the more the symptoms resemble those of cancer of the sigmoid. Diarrhea may at times supervene; occasionally after sloughing of the tumor, paralysis of the sphincter occurs.

Leube was the first to call attention to the fact that *hemorrhoids frequently developed with carcinoma of the rectum*, and that, too, at an early stage. Piles *occurring suddenly*, and not developing slowly or existing for many years, are suggestive.

Rectal examination is most important, and the finger will usually give the necessary information. One can feel a mass lying directly beneath the mucous membrane or adherent to it. The surface may feel uneven and hard or there may be occasionally a cauliflower or mushroom growth, or a constriction, through which the finger cannot readily pass. If it be barely possible to feel the growth, it is similar in sensation to the cervix uteri.

Blood, pus, or sanious material may be found on the finger if the growth is ulcerating.

Vaginal examination in women must be made to differentiate the source of the tumor. The genito-urinary organs in the male should also be examined.

Simple cicatricial stricture is usually *smooth* and not ulcerated, while a carcinomatous stricture is usually nodular and frequently ulcerated.

It is preferable for accurate diagnosis to excise a portion under cocain for microscopic examination.

If the stricture cannot be reached by the finger, Kelly's speculum is of value. *Never* insert the hand into the rectum.

Metastatic growths in other organs are quite frequent from small growths of the rectum; thus, with carcinoma of the liver, a small primary carcinoma of the rectum may be found, even if no symptoms are present.

The bladder and vagina may be involved and fistulous openings occur. Periproctitis (abscess) and fistulæ are rare.

The peritoneum is rarely involved unless the carcinoma is high up.

Course.—The termination is death if not operated on. This may occur by occlusion of the intestines and from peritonitis.

It is hardly possible to give a prognosis as to duration. In cancer of the duodenum the general nutrition suffers early, or the case may be very acute and the duration of life is short. In many cases the course varies from six months to two years, while in rectal cancer it may last for three to even four years. Rarely cases come to a standstill and last some years.

In some cases coma (carcinomatosum) appears quite early, probably due to auto-intoxication from intestinal decomposition or from the toxins of cancer. Ewald isolated a body from the urine belonging to the group of diamins in such a case.

Thrombosis may develop and embolus of the lungs occur, with death resulting. Death may occur from exhaustion.

Diagnosis.—Presence of a tumor by abdominal palpation or rectal examination, accompanied by cachexia (loss of weight marked) and anemia, with marked constipation and increasing symptoms of stenosis of the bowel; or cachexia, intestinal disturbances, with no detectable tumor, but with symptoms of progressive stenosis in an elderly person, are suggestive of cancer. Examination of a tumor fragment, if it can be secured in the stool or from the rectum, is conclusive.

Prognosis.—This is fatal, unless early and complete removal.

Treatment.—Complete and early removal of the growth is indicated. An early diagnosis is important. If abdominal cancer is suspected, *exploratory laparotomy* and complete resection with end-to-end or lateral anastomosis are indicated.

If resection is impossible, entero-enterostomy or enterocolostomy for drainage to relieve symptoms.

If the tumor is low down in the colon, sigmoid, or rectum and inoperable, then colostomy to relieve the symptoms and prevent irritation of the surface of the cancer.

In the rectum, resection, preferably Kraske's operation, is indicated if radical operation is possible.

Palliative curetment and the thermocautery may be employed in some cases. Colostomy under cocain can be performed in the aged and feeble.

Trypsin treatment, described under Gastric Cancer, is *justifiable* in inoperable cases, or in those in which a palliative operation has been performed. Coley's treatment by erysipelas toxins may be tried, but I would not recommend it. It is more successful with sarcoma.

Diet.—Soluble foods with little residue, such as milk, broth, bouillon, sanatogen, somatose, cream, butter, rice-gruel, sour milks, matzoon, kumyss, bacillac, etc., are indicated.

Irrigation of the intestines, enemata, and injections with olive oil, and internally, castor oil, magnesium sulphate, rhubarb, cascara, etc., to keep movements soft; warm applications to the abdomen; morphin, codein, and belladonna are indicated for pain. These last can be given by suppository. Strength should be supported by tonics, iron, arsenic, etc., and pain relieved. Treat complications.

SARCOMA AND LYMPHOSARCOMA OF THE INTESTINES

Sarcoma of the intestines is rather an infrequent disease, much less frequent than carcinoma. In the course of twelve years, 1882–93, in the Vienna General Hospital, out of 274 autopsies on sarcoma, only 3 were sarcoma of the intestines, the ileum, cecum, and rectum. In 61 cases of lymphosarcoma, 9 belonged to the intestines, 1 in the duodenum, 3 in the jejunum, 3 in the ileum, and 2 in the cecum.

Of Libmann's cases, 15 were of the duodenum, 18 of the jejunum and ileum, 14 of the ileum, and 3 of the entire small intestine.

Sarcoma occurs as frequently in the small as in the large intestine, and *lymphosarcoma preponderates* in the small bowel,

Of Krueger's 37 cases, 16 were of the small bowel alone and 16 of the rectum; the ileocecal region comes next.

Sarcomata usually attain a large size, even as large as a child's head, and spread over a large part of the intestines.

Anatomy.—*Sarcomata usually originate in the submucosa*, the musculature is attacked early, and the serosa is rarely involved. Small round-celled sarcoma is most frequent; occasionally spindle celled. In the rectum they are often melanotic.

Lymphosarcomata start from the lymphatic apparatus, the solitary and agminate lymph-follicles. These occur chiefly in the small intestine.

Sarcomata of the intestines show a rapid progress and metastases are found early. *They do not produce stricture of the bowel*, but a dilatation; and develop in a longitudinal direction along the intestines. The bowel may be enormously dilated. Rectal sarcoma, however, may produce obstruction.

Age.—Sarcoma is frequent in young persons; most frequent from twenty to forty years of age; from four to seventy years cases are reported, and one congenital case in an infant three days old.

Symptoms.—The clinical symptoms differ from carcinoma. The general health becomes impaired early. The patients emaciate rapidly and become anemic. There is a peculiar want of proportion between the rapid impairment of the physical condition and the absence of local symptoms. They become rapidly weak and debilitated (cachexia). There are, as a rule, an absence of abdominal pain and no symptoms of stenosis. Intestinal symptoms are slight—occasionally constipation, alternating with diarrhea.

Exceptionally, stenotic symptoms appear, due to kinking .or peritonitic incarceration, rarely ileus. Stenosis occurs in rectal carcinoma.

The tumor appears early, grows rapidly, and can easily be made out.

Duration.—The majority of cases die within nine months from general cachexia; only 1 case recorded lived twenty-one months.

Prognosis.—This type of tumor proves fatal, and even early operation seems of no value on account of the metastases.

Treatment.—Injection of Coley's fluid, the mixed toxins of erysipelas and Bacillus prodigiosus, is indicated, as in inoperable sarcoma in the other regions. One must remember that marked reaction follows the injection in some cases, and some patients cannot undergo the treatment. I have seen the latter occur, and also in another case temporary improvement.

The patient should receive as liberal a diet as possible, and iron, arsenic, and cod-liver oil should be administered. As a rule no stenosis is present, but if in evidence, operative procedure is indicated. Removal has failed on account of metastases.

BENIGN TUMORS OF THE INTESTINES (NEOPLASMS)

Benignant neoplasms of the intestines are relatively rare, and their clinical significance is generally slight. They occasionally produce severe symptoms.

These tumors may be attached to the bowel wall by a broad base or by a thin pedicle or stem, and are then termed polypi. The last type is usually of small size, that of a cherry, but rarely as large as a pear, and occur most frequently in the rectum.

The following forms of benign tumor are found: Adenoma, fibroma, lipoma, papilloma, myoma, fibromyoma, angioma, and chylangioma.

Adenomata are most frequently met with. They arise from the glands of Lieberkühn and in the duodenum from Brunner's glands; are acinous in structure, and may be attached to the bowel by a broad base or by a pedicle. As a rule they are small, the size of a pea, rarely the size of a pear. They are red and have a tendency to bleed; usually they are soft and the surface is smooth, though occasionally of a cauliflower appearance. They occur most frequently in the rectum, are usually polypoid, though occasionally annular and flat. They are most common in children from four to seven years, though occasionally in adults. More than half the cases that occur are in males between sixteen and thirty years of age. Sometimes there is extensive involvement of the intestines, a condition known as polyposis intestinalis adenomatosa; in 1 case several thousand were present. Polypi are never limited to the small intestine. Carcinomatous degeneration of the polypi may occur.

Fibroma, Lipoma, Papilloma.—These neoplasms derived from the connective tissues, especially from the submucosa, are very rare. They are, as a rule, lipomatous in structure, and often originate from the appendices epiploicæ. They frequently project into the peritoneal cavity and may twist their pedicle and become detached. They may have a broad base or pedicle, are of various shapes, and may grow to the size of an apple. They occur most frequently in the colon and rectum, less frequently in the jejunum, and rarely in the ileum.

Myoma, Fibromyoma.—These are very rare and usually originate from the outer muscular coat of the intestines, chiefly from the longitudinal coat. There are two forms: Spheric nodules, which grow toward the mucous lining and sometimes become pedunculated, the mucous membrane lying loose in front; or they may have a broad thick base, forming a circumscribed thickening, the mucous membrane being adherent.

Rarely the myoma will grow toward the peritoneal cavity.

Angiomata.—These are exceedingly rare, and may occur as a telangiectatic mass, or as a flat vascular tumor involving more or less of the bowel wall. Cystic chylangiomata occur in the small intestine.

Symptoms.—In many cases there are no symptoms at all, and the condition is discovered accidentally post mortem.

Sometimes they give rise to intestinal hemorrhage. When this occurs in a person in good health, who has never given any history of previous intestinal trouble, the possibility of an intestinal tumor should be thought of. This is especially true in children.

Diarrhea with blood and mucus have been reported in cases of polyposis intestinalis adenomatosa. On account of their small size it is nearly impossible to discover them by palpation.

Symptoms may rarely be produced by narrowing or occlusion of the bowel by the intestinal tumor, or from an intussusception caused by the tumor. When tumors are situated in the rectum disturbances are most likely to occur, such as hemorrhage, passage of mucus, tenesmus, and difficult defecation.

Sometimes the mass may be felt by the examining finger, and it may even protrude from the anus and give rise to severe pain. It may occasionally be torn off and passed with the stools, when the symptoms may disappear.

Course.—They may remain latent or give symptoms for some years and then be passed per rectum.

Treatment.—Intestinal hemorrhage should be treated in the manner already described under Typhoid. The blood will often be bright in color if the hemorrhage is from the colon or rectum; injection of very hot or cold water with ʒj (4.0) of alum or tannic acid, gr. 30 (2.0), locally, or Trémolière's solution, by mouth and rectum; ice-bag to the abdomen; morphin, gr. ¼ (0.016), and ernutin, ♏v (0.296), by hypodermic, are indicated.

If the tumors are accessible in the rectum, they should be removed by galvanocautery or by operation.

GAS CYSTS OF THE INTESTINES

(*Synonym.*—Pneumatosis Cystoides Intestinorum Hominis.)

Cysts containing air, in the intestines of pigs, were probably first described by Mayer,[1] of Bonn. This condition was also independently described by John Hunter. These intestinal gas cysts have been found quite frequently in apparently otherwise healthy pigs and occasionally in sheep. The theory has been held that bacteria are the cause, or that they are the result of mechanical and physical conditions; for example, that gas may escape through some abrasion into the tissues. Bang[2] first reported this condition in the human being, and Finney the first case in America.

Finney and Welch[3] believe the cyst to be a distinct variety of tumor, the cells of which have the faculty of secreting gas. In practically every case so far the gaseous cyst has been associated with disease of some portion of the intestinal canal, producing an obstruction to the lumen of the bowel. Cysts may be single or multiple and in the small and large intestines.

Pathology.—There is a dense fibrous tissue framework containing round and spindle cells, and there are clefts and spaces whose walls contain large giant cells with many nuclei. Air spaces are found about these cells, and there is an endothelial lining to these spaces. The blood-supply is rich and hemorrhages may occur in the tissues. Air in the cyst resembles atmospheric air. The tumor is more pronounced in the subserous tissue, though it has been observed in all the layers of the walls.

Clinically, there is no definite picture. Crepitation on palpation has been noted. Pain and constipation could be chiefly attributed to other factors.

Diagnosis is usually made during operation for some other trouble or at autopsy.

Treatment.—If symptoms of obstruction, operation is indicated. After operation for other conditions, they generally diminish in size or disappear.

[1] Jour. d. prakt. Heilk., 1825.
[2] Nord. med. Ark., 1876, viii, No. 18.
[3] Jour. Am. Med. Assoc., October 17, 1908.

CHAPTER XXVIII

HEMORRHOIDS; PROLAPSE OF RECTUM; FISSURE

HEMORRHOIDS

(*Synonyms.*—Phlebectasia Hemorrhoidalis; Piles.)

Hemorrhoids consist of diffuse or circumscribed varicose dilatations of the hemorrhoidal veins, lying either in the subcutaneous tissue of the anus external to the sphincter (external hemorrhoids) or in the submucous tissue of the lower portion of the rectum (internal hemorrhoids).

The hemorrhoidal veins surround the lower portion of the rectum and *there form the hemorrhoidal plexus.* The majority of these veins enter into the inferior or external inferior hemorrhoidal veins, and from there into the common pudic and iliac veins; others pass into the median inferior hemorrhoidal veins and so into the internal iliac vein and the inferior vena cava. A small number of the veins enter the superior hemorrhoidal veins, thence into the inferior mesenteric veins and the portal system.

In portal obstruction blood from the hemorrhoidal plexus can pass into the vena cava; while back pressure on the vena cava inferior, as of cardiac origin, may affect the hemorrhoidal system. All hemorrhoidal veins *are devoid of valves.*

Anatomy.—External hemorrhoids are visible to the naked eye, lie below the sphincter ani, and are often arranged in groups around the anal orifice. They appear as bluish-red, tortuous vessels encircling the opening; or there may be isolated varicose protrusions, from the size of a pea to a walnut. They may be round, flat, or irregular in shape and their size sometimes changes in the same patient, often being smaller after defecation.

Internal hemorrhoids often can only be discovered by digital examination or by use of the proctoscope, as they lie above the sphincter. In aggravated cases the patient by bearing down may cause them to protrude. They usually appear as soft nodules of bluish hue, with thin walls.

The diffuse or circumscribed nodular forms may be present, and the last constitute a true varix.

External and internal hemorrhoids may occur together. Piles may occur singly or in pairs, or be multiple and form a ring about the anal opening, both external and internal to the sphincter, and even a third higher ring has been described. In exceptional cases dilated veins are found high up the rectum even into the sigmoid flexure.

records. Habitual constipation, pelvic congestion, and sedentary life favor markedly the production of hemorrhoids, and these conditions we find most frequent in women. *Males will at once consult the physician* if there is difficulty in the anal region. Women are, as a rule, extremely sensitive and usually treat their piles by home remedies until conditions are such as to necessitate advice from the physician. It has been my experience that most of my women patients never refer to "hemorrhoids" unless specifically questioned. Actually I believe women are the more frequently afflicted.

Age.—Piles occur most frequently between thirty and fifty years of age. The modern consensus of opinion is that hemorrhoids are a local disease and not due to any diathesis or faulty state of the general circulation. There are certain peculiarities in the rectal plexus of veins favoring the production of piles which are as follows:

The walls of the hemorrhoidal veins are thin and contain few muscular fibers, and hence their contractile force is less than that of the veins in the lower limbs.

There are no valves in the rectal veins, so that blood can be readily forced back into them. Muscular contraction aids the onward propulsion of the blood in other regions.

In the rectum fecal masses compress the blood-vessels, the sphincteric contractions compress the vessels, and contraction of the abdominal muscles during defecation interfere with the return of blood. They are dependent blood-vessels, and with the unfavorable factors mentioned it is not surprising that hemorrhoids are frequent.

Some of the hemorrhoidal veins enter the portal system, in which there is a low degree of pressure and in which the circulation is readily retarded. Stasis of this system may be a contributory factor in the production of hemorrhoids, as in cirrhosis of the liver; as may also diseases of the heart and lungs leading to engorgement of the venous system.

All conditions which tend to produce interference with the return circulation of blood from the hemorrhoidal vessels, or produce or predispose to hyperemia, influence the production of hemorrhoids. Among such are: chronic constipation; sedentary habits, as in clerks, students, shoemakers, seamstresses, sitting on soft cushions, excessive horseback riding, etc.; enlarged uterus, as from disease or pregnancy; prostatic affections, tumors of the bladder or growths in the pelvis.

Hemorrhoids may develop suddenly in cases of carcinomatous stricture of the rectum and early in the course of the disease.

Habitual constipation favors the production of piles. Hemorrhoids also may be the primary condition, and on account of the pain of defecation the patient avoids having a movement as long as possible. Excessive venery is given as causing congestion in these regions. Abuse of alcohol, spiced food, fatty food, excessive quantities of food and drink are believed by some to favor the plethoric habit and

Usually, subjective symptoms are present, such as itching, burning, pressure and weight in the anus, and the sensation of a foreign body, with tenesmus. There may be feelings of pressure and even pain in the sacral and lumbar regions. Occasionally soreness over the lower part of the abdomen, pressure in the rectum, bladder, uterus, and vagina; pains radiating to the thighs may be present. Sometimes these symptoms precede a hemorrhage, after which temporary relief occurs.

As a rule, the bowels are irregular and constipated.

Hemorrhages may be frequent, so as to cause marked secondary anemia. Bleeding may be severe and bright red blood gush from the rectum nearly pure, or it may be dark brown mixed with fecal matter. In such an event it lies on the surface of the feces and is not intimately mixed with it. Small quantities of blood are frequently passed with hard stools.

Hemorrhages sometimes occur on no special provocation, or after a horseback or bicycle ride, etc.

Gummy acrid mucus may be discharged from the hemorrhoids due to catarrh of the rectum, and may cause excoriations.

In some the hemorrhage may last for several days and temporary relief may occur. It has been stated that bleeding from hemorrhoids may take the place of menstruation (vicarious).

Dyspnea, palpitation, angina, hiccough, giddiness, despondency, and hypochondria, often attributed to piles, I believe, with Riegel, are merely associated with the constipation present.

Proctitis, fissure, and occasionally abscesses or fistulæ may develop.

Disturbances of adjacent organs, such as strangury, hemorrhage from the bladder or vagina, or catarrh of the latter may occasionally occur.

Diagnosis is made by inspection and digital examination; occasionally the proctoscope is necessary. The appearance of hemorrhoids has been described.

Condylomata encircle the anus and are often present on the scrotum. There are a history of syphilis and manifestations of lues.

Skin tags are whitish looking, do not change their size, and do not bleed when punctured. Piles differ in the last regard.

Internal hemorrhoids are reddish blue and bleed when punctured; polypi do not do so. The latter occur most frequently in children.

Carcinoma presents a hard consistency, stenotic symptoms, and cachexia. A small section placed under the microscope will settle the diagnosis.

Piles may occur suddenly in association with cancer.

Prognosis.—Piles are frequently a chronic affection and may exist throughout life. In cases occurring during pregnancy, restitution to normal is possible. This is true of the milder types if the cause is corrected. They may markedly diminish in size and then an exacerbation take place. They rarely endanger life unless strangulation with gangrene or a large abscess or dangerous hemorrhage occur.

Treatment.—Excesses of all kinds in food, drink, and venery should be avoided. The patient should lead an out-of-door life, take proper exercise, and have a daily soft evacuation of the bowels.

All conditions causing venous engorgement of the rectum should be avoided, such as constant standing or sitting, horseback and bicycle riding. The patient should not sit on warm soft cushions, but on cane-seats or those covered with leather or horse-hair.

Diet.—The diet should be mixed in character and in part consist of a considerable variety of green vegetables and raw ripe and cooked fruits, to regulate the bowels.

It should suit the individual, and a stout full-blooded person must be somewhat limited in his diet. The patient should not take three large meals at long intervals, but preferably smaller and more frequent feedings.

Alcoholic beverages, strong coffee, strong tea, highly seasoned dishes, cheese, coarse brown bread, peas, corn, and baked or lima beans are best avoided.

String beans, spinach, asparagus, and green salads aid bowel action. Potatoes, rice, beets, and cauliflower may be allowed in small quantity. Raw fruits, such as apples, pears, prunes, grapes, oranges, and stewed fruits, such as prunes, baked apples, etc., are of value. Soups of various kinds are allowed. Hot breads, richly spiced foods, and rich desserts should be avoided.

Matzoon, kumyss, bacillac, lactone-milk, and buttermilk often have an excellent effect on the bowels. They can be taken as the extra meals midway between the usual meals. A few crackers or zwieback with plenty of butter can be given. Some recommend water only between meals and interdict much of that. Unquestionably a certain amount of fluid with the food is an aid to digestion and bowel action. I have seen severe cases of constipation result from the elimination of all fluids at meals.

At least ℥viij (250 cc.) of fluid in the form of broth, soup, cocoa, or water should be taken at each meal. On rising, administration of a glass of water often aids bowel action, and between meals an equal amount should be given, or matzoon or kumyss substituted.

Outdoor exercise, such as walking, is of service. It should not be carried to the point of fatigue and is contra-indicated during acute inflammation of the piles or if hemorrhage or unpleasant symptoms result. Massage, gymnastics, and Swedish movements are of value.

Bowels.—Constipation should receive appropriate treatment. *Powerful cathartics should be avoided.* The dietary methods mentioned should be carried out. The patient should go to stool daily at a definite hour. I have found the administration of olive oil, 1 to 2 tablespoonfuls (15.0–30.0) t. i. d. before meals, an aid to bowel action. The injection of ℥j to ij (30.0–60.0) of olive oil into the rectum with a small soft-rubber ear syringe just before having or attempting to have a movement is a valuable procedure. It renders the passage easier and less painful.

Aloes should be avoided in hemorrhoid cases.

Among our simple remedies are compound liquorice powder, ʒj (4.0); fluidextract of cascara or aromatic fluidextract, ʒj (4.0); extract of cascara, gr. 1 to 5 (0.06–0.3); purgin (phenolphthalein), gr. 1½ to 5 (0.1–0.3); tincture of rhubarb, ʒj (4.0), or extract of rhei, gr. 4 (0.25). Other remedies are described in the chapter on Constipation.

Carlsbad, Kissengen, and Saratoga waters are of service, especially at the springs.

A good prescription is the following:

℞. Ext. cascara..............................gr. xv (1.0)
Ext. belladonnæ }
Ext. nucis vomicæ }....................āā gr. iij (0.2).—M.
Make 12 pills.
Sig.—One to two at bedtime.

Hygiene of the Anus.—After defecation, cleansing with a moist sponge or wet cotton is less irritating than paper.

External piles should be protected with a small piece of cotton moistened with sweet oil or covered on the anal side with vaselin, boric acid, or zinc ointment. Cold sitz-baths and cold ablutions to the rectum are valuable. An external douche with cold water is excellent, thus: A fountain syringe is filled with cold water at about 60° F. and several ounces of witch-hazel added, or a hot douche at 110° to 115° F. can be substituted. The rectal tip is placed close to the anus, which is then thoroughly douched. This is especially applicable for external piles, and lessens congestion even when they are internal.

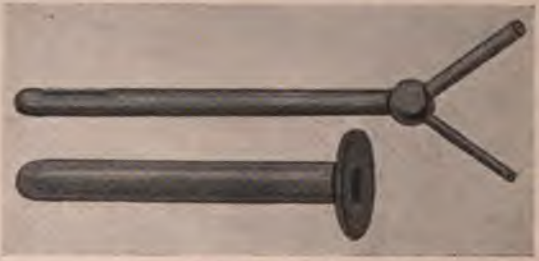

Fig. 213.—Kemp's tube (cooler) for hemorrhoids or prostate.

Fig. 214.—Kemp's ice tube for hemorrhoids.

For external inflamed piles stronger medicated local applications can be employed than with internal piles, where greater absorption occurs.

External Hemorrhoids.—With inflamed piles absolute rest in bed or on a couch should be enjoined. A small ice-bag or gauze wrung out in cold solution of witch-hazel and water, equal parts, or of lead-and-opium wash, are excellent applications. Some prefer the application of warmth; cold is usually more efficacious.

For the application of cold to relieve congestion, both to external and internal hemorrhoids, the simple instruments in Figs. 213 and 214 are of value.

The ice tubes for hemorrhoids are made in a nest containing several sizes, in appearance like a very small test-tube with a broad flange, to prevent slipping into the bowel. The tube is filled with powdered ice, corked, well lubricated, and inserted into the anus. They are of glass or metal, the latter the safer.

The prostatic cooler is of small caliber—a simple closed tube with a large entering and small return attachment. The fountain syringe is attached to the large branch and a soft outflow tube to the smaller. Very cold (preferably) or hot water can be employed. The soft-rubber rectal bag (Fig. 215) is made on similar principles, but is more bulky.[1] They were reported some years ago.[2]

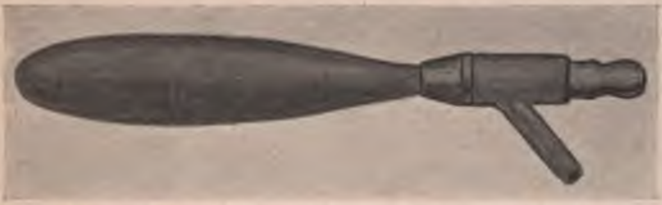

Fig. 215.—Kemp's soft-rubber rectal bag, used as a cooler for hemorrhoids.

In addition, one can attach a small catheter to the tip of the fountain syringe and, inserting the soft tube about 1½ inches, thus douche the internal hemorrhoids. The current should be slow, the patient evacuating the fluid around the catheter every minute or so. Hot or cold normal saline solution with witch-hazel, ℥i (30.0) to the pint (500 cc.), can be employed. The old Bodenhamer recurrent tip or the flexible or hard-rubber recurrent tubes (Kemp) can be used, introducing them only about 2 inches; but with inflamed piles they are apt to cause more pain than the small soft catheter.

I have found the following prescriptions, recommended by Samuel Gant, of value for the relief of pain and inflammation in external hemorrhoids:

℞.	Liq. plumbi subacet.	ʒiv	15	
	Tinct. opii	ʒiiss	10	
	Aq. destil.	q. s. ℥iv	125	.—M.

Sig.—Apply cold, on gauze.

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[1] A small rubber condom, with two catheters tied in, one for the entering and the other for the outflow current, can be substituted for this bag.

[2] Manual on Enteroclysis, Hypodermoclysis, and Infusion, 1900.

℞.	Ung. stramonii	ʒiss	6	
	Ung. belladonnæ	ʒiiss	10	
	Ung. acidi tannici	℥ss	15	.—M.

or

℞.	Morph. sulph	gr. iij		.195
	Cocainæ hydrochlor	gr. xij		.78
	Vaselin	℥ij	60	.—M.

or

℞.	Cocainæ hydrochlor	gr. v		.325
	Ext. bellad. Ext. opii Ext. aconite Ext. stramonii	āā ʒij	8	
	Glycerin	ʒss	2	.—M.

Sig.—Apply on cotton, externally.

This last prescription I prefer to use first at about one-third or one-half strength. It should be employed only for the temporary relief of severe pain and should be left on only for a short period, about fifteen minutes.

Cold or, at times, hot injections, and opium and belladonna suppositories are excellent for tenesmus.

For internal hemorrhoids, when prolapsed, inflamed, and difficult of reduction, it is advisable to apply cold, such as ice-water to which witch-hazel has been added, ice, or lead-and-opium lotion (cold), for a brief period to lessen edema and inflammation.

Local *application of adrenalin* (1:1000) *to an irreducible pile* lessens congestion and aids reduction.

One of the following ointments is excellent as a sedative:

℞.	Morph. sulph	gr. viij		.52
	Hydrarg. chlor. mit	gr. xij		.78
	Vaselin	℥j	30	.—M. (Gant.)

℞.	Ext. opii	ʒss	2	
	Cocainæ hydrochlor	gr. x		.65
	Mentholi	gr. xx	1	.30
	Ung. zinci oxidi	℥j	30	.—M.

The prolapsed hemorrhoid should be well lubricated before the physician endeavors to push it up with the finger. Occasionally an anesthetic may have to be administered. There are the dangers of strangulation with gangrene and sepsis from an unreduced hemorrhoid.

For ulcerated hemorrhoids the following are useful:

℞.	Bismuth. subnit	ʒij	8	
	Hydrarg. chlor. mit	℈ij	2	.6
	Morph. sulph	gr. iij		.195
	Glycerin	ʒij	8	
	Vaselin	℥j	30	.—M.

Sig.—Apply with pile syringe (Allingham).

℞.	Cocainæ mur	gr. xij		.78
	Iodoformi	℥j	4	
	Ext. opii	ʒss	2	
	Vaselin	℥j	30	.—M.

Sig.—Apply with pile syringe (Mathews).

For hemorrhage the following are of service: Rectal injections of ice-water, ℥viij (250 cc.), alone or with tannic acid, gr. 5 (0.3), dissolved therein, or alum, ʒj (4.0), or witch-hazel, equal parts; local application of ice-bags or the ice tube.

Several ounces of Trémoliére's solution—gelatin (5 per cent.) with chlorid of calcium (2 per cent.)—can be injected into the rectum. It was originally used as a local styptic.

If the bleeding is from external piles, styptics can be applied in ointment form; if from internal hemorrhoids, the ointment can be inserted with the finger or applicator, or as a suppository.

Among such useful remedies are unguentum acidi tannici and unguentum gallæ:

℞.	Unguentum acidi tannici	ʒiv	16	
	Unguentum stramon. Unguentum belladonnæ	āā ℥j	30	.—M.

Ft. ung.

℞.	Suprarenal ext.	gr. v		3
	Ol. theob.	gr. xxx	2	.—M.

Ft. suppos. No. i.

℞.	Adrenalin chlorid (1: 1000)	♏x (.592)
	Ol. theob.	q. s.—M.

One suppository.

℞.	Ichthyol, Acidi tannici	āā gr. v		3
	Ext. belladonnæ Ext. stramon.	āā gr. ⅓		022
	Ext. hamamelis	gr. x		6
	Ol. theob.	q. s.		.—M.

Ft. one suppository. (J. P. Tuttle.)

An ointment or suppository containing chrysarobin has been advised for the treatment of hemorrhage from piles:

℞.	Chrysarobin	gr. xv	1	
	Ext. belladonnæ	gr. v		3
	Iodoform	gr. x		6
	Petrolati	ʒss	15	.—M.

Ft. unguentum.

℞.	Chrysarobin	gr. ij		13
	Acidi tannici	gr. iij		194
	Iodoformi	gr. ij		13
	Ol. theob.	gr. xxx	2	.—M.

Ft. one suppository.

In many cases of hemorrhage the simpler remedies are sufficient. Rarely it may be necessary to tampon the rectum. This can be performed by slipping in a piece of cheesecloth like the finger of a glove, in effect, a bag, with the outside well lubricated. This is packed with cotton tampons or strips of gauze, and the distended bag drawn down against the sphincter. It can be done in emergency without an anesthetic. If these measures fail, it may be necessary to ligate the bleeding artery or, if this be impossible, then the entire hemorrhoid.

Radical Treatment.—*Dilatation of the Sphincters.*—By means of this the spasm of the sphincter is stopped, the pressure on the blood-vessels is relieved, and the bowels act more easily. It will frequently be of benefit in the early stages of hemorrhoids, and I have seen it relieve severe symptoms in advanced cases, especially if fissure is associated. The author has noted cases in which several years after dilatation the patient has claimed to be in comfort, with no recurrence. As a palliative the procedure is justifiable, especially if fissure is present.

Gradual dilatation by the use of specula of increasing size or of dilators can be carried out. The procedure takes several weeks and is necessarily very painful. If this method is carried out, air dilatation with Roberts' dilator (Fig. 216), made on the principle of Barnes' cervical dilator bag, is the most satisfactory. The hard-rubber dilators cause more pain.

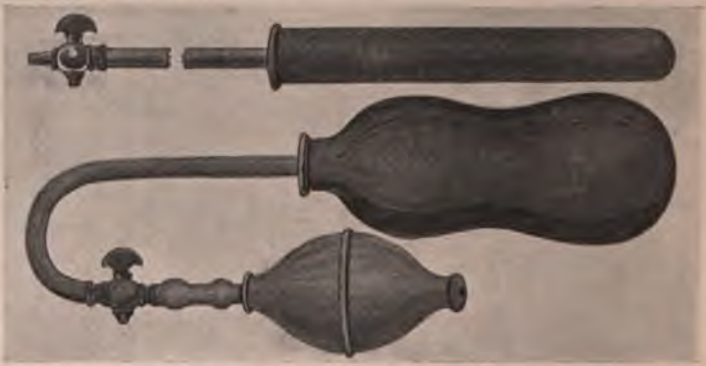

Fig. 216.—Roberts' dilator.

Rapid and complete dilatation under anesthesia is the best method when safe for the patient. With the aid of a skilled anesthetist it can be satisfactorily performed under nitrous oxid. Other anesthetics can be employed. The thumbs must be inserted into the rectum and the sphincter *dilated gradually,* both laterally and anteroposteriorly. All spasm must be overcome and the sphincter thoroughly relaxed. Violence must not be employed. An opium suppository, gr. 1 (0.065), with belladonna, gr. 1/3 (0.02), should be inserted and the bowels kept closed for about forty-eight hours.

Carbolic Acid Injection.—This method has been recommended in order to produce shrinking of the piles and so avoid radical operation. It should not be performed if the hemorrhoids are inflamed.

The following solutions for injection have been recommended:

℞.	Acid. carbolic. (Calvert's)	ʒij	8
	Acid. salicylic.	ʒss	2
	Sod. bibor.	ʒj	4
	Glycerin (sterile)	q. s. ℥j	30.—M.

(J. P. Tuttle.)

The solution should be syrupy and clear; if white, it is imperfect.

The average injection into a hemorrhoid is ♏v (0.296). Not more than ♏x (0.592) should be injected into two to three hemorrhoids. After the injection a suppository of—

℞.	Opii (pulv.)	gr. j	065
	Ext. belladonnæ	gr. ⅓	022
	Iodoformi	gr. ij	13
	Ol. theob.	q. s.	.—M.

Carbolic acid and glycerin, equal parts; or carbolic, 1; glycerin, 3; water (distilled), 3, have been employed.

Inject 3 to 5 drops with a hypodermic syringe into the center of the pile. Care must be taken that none of the solution drops from the needle and cauterizes the mucous membrane. An enema is first given, the piles cleansed and dried, and then anointed with iodoform ointment after injection with carbolic.

A few drops of a 1 per cent. cocain solution may be first used subcutaneously to deaden the pain.

It is preferable to inject one hemorrhoid at a time. Considerable pain at times follows. The possible dangers are ulcer, abscess, and fistulæ. There is some danger of sepsis and cases have been reported.

Cauterization with Fuming Nitric Acid.—The surrounding parts are covered with a thick layer of vaselin except the pile that is to be treated. This is painted with the nitric acid by means of a small stick or glass rod. Care must be taken that the acid touches no other spot. The pile turns a grayish-green color as a result. Iodoform ointment or vaselin is applied and the hemorrhoid pushed back into the rectum. Allingham advises carbolic acid as a substitute for the nitric acid. There is an element of danger from sepsis in this method.

Ligature of hemorrhoids under anesthesia without removal and then allowing them to *slough off* has been employed. The method is not to be recommended for obvious reasons.

Crushing.—The hemorrhoid is crushed longitudinally by means of a special instrument, the projecting portion *cut off*, and pressure kept up for about a minute. This technic is not advised.

Clamp and Cautery.—Each pile is seized with a volsellum forceps and drawn well down. The clamp is applied to embrace its base. The portion above the clamp is cut off with scissors, and the cautery iron, heated to a dull red, is applied to the stump until the vessels are well seared. Either the thermocautery or galvanocautery can be employed. Excellent results have been secured by this operation.

Ligature and Extirpation (Allingham's Operation).—Incision is performed at the base of each pile through the mucosa, and the pedicle dissected out. This is ligatured and the pile cut off with scissors. Some operators remove every alternate hemorrhoid, the rest usually shrinking as a result of operation. Others incise the mucous membrane above the base of the pedicle and force it back like a cuff. After ligature and removal of the pile, the cuff of mucosa is drawn down and a single stitch taken. This last method is to be recommended.

Extirpation and Suture (Whitehead's Operation).—The mucous membrane containing the hemorrhoids is dissected out, the incision frequently encircling the anus. The piles are removed, and the mucosa, being loosened up, is drawn down and attached by sutures to the skin at the anal margin. *Stricture* or incontinence of feces are sequels which may follow this operation. Subsequent treatment by bougies is necessary if stricturing be feared or if it occur.

In these operations anesthesia is necessary, and dilatation of the sphincter previous to removal of the hemorrhoids.

It is usually customary to close the bowels for twenty-four to forty-eight hours after operation by means of opium suppositories, though Graser opens the bowels at once.

Gant's Water Infiltration Method.—This method, devised by Samuel Gant, is of special value in cases where an anesthetic is contraindicated or when the patient objects to its employment. With a large hypodermic sterile water is injected about the base of each hemorrhoid in the submucosa, producing thorough infiltration. An incision is then made around the base of the pile, the pedicle dissected out, the latter ligatured, and the pile cut off with scissors.

Careful preliminary dilatation of the sphincter is carried out.

Having seen this operation performed by its originator and also the ultimate results, I can recommend it as excellent.

Complications of Hemorrhoids.—*Prolapse of the Rectum.*—*Etiology.*—Rectal prolapse is a fairly frequent complication of hemorrhoids, though it may occur alone. It may involve the mucous membrane alone or all the coats of the rectum (procidentia recti). Among other causes of prolapse are polypi, vegetations, tumors; conditions tending to weaken the sphincters, such as ulceration or operative incisions, spinal paralysis, or traumatism; conditions producing muscular spasm, such as worms, dysentery, phimosis, cystitis, calculus, enlarged prostate, or stricture of the urethra.

In some cases with hemorrhoids, the prolapse may only be partial as regards the circumference of the rectum, while in others it involves the entire circumference and there is a scarlet or livid mass projecting from the anus. An internal prolapse of the rectum may occur, in which the upper part descends through the lower part, but does not appear outside the anus. It is probably due to relaxation of the ligaments of the rectum. It corresponds rather to an intussusception.

Weakness and paralysis of the sphincters are predisposing factors.

Prolapse of the rectum is frequent in debilitated children, especially if there is intestinal catarrh with a tendency to frequent stools accompanied by straining. Rectal prolapse occurs in elderly people, also in cases of severe constipation, and as a result of frequent pregnancies.

Symptoms.—With moderate prolapse, during the act of defecation, a protrusion of the rectum from 1 to 2 inches long appears outside the anus. It is red or bluish red in color and puckered in appearance, covered with some mucus. The swelling is continuous, with the skin on one side and with the mucous membrane on the other, and is arranged in folds which radiate from the central aperture toward the circumference.

In advanced cases the mass resembles a tumor with a star-like opening at its center, and the color is a paler red or bluish. With children the mass generally protrudes only when at stool; while with adults it comes down more readily or often remains constantly down. At first it is spontaneously reducible, later, easily replaced by slight pressure, and finally it may become very difficult or nearly impossible of reduction.

It often becomes inflamed or ulcerated, and in old cases incontinence of feces may occur.

At first there is no or little pain, but after a time it may become quite severe.

There is a tendency for the prolapse to increase in size. If inflammation occur, there will be fever and constitutional disturbances.

As a rule, there is no marked bleeding from the prolapse itself, but more of an oozing. Hemorrhage from associated hemorrhoids may occur.

Diagnosis.—This is made from the appearances described. If operation is contemplated, it is important to differentiate between prolapse of the mucosa alone or of the rectum. In the latter event the peritoneum may be involved and the intestines be found in the prolapsed portion.

Prolapse of the mucosa is not as firm and thick to the feel, the folds of the mucous membrane radiate from the orifice to the circumference, and the opening is circular and patulous.

With prolapse of the entire wall the tumor is more conic, the walls are thick and firm, the orifice is slit-like. When the mass is pressed between the fingers gurgling of gas in a contained loop of gut may sometimes be heard and resonance obtained on percussion.

In some cases where there is much inflammatory thickening it is difficult to differentiate. In following out treatment this possible danger should be recognized and due precaution be exercised.

Internal prolapse is more difficult to recognize. Digital examination of the rectum is necessary. First keep the finger close to the anterior wall until it passes into a cul-de-sac. Withdraw it slightly and examine the center of the mass until an orifice is found into which the finger or a bougie can be passed for some inches into the rectum.

It may be necessary for the patient to bear down during examination.

Treatment.—Predisposing factors should be eliminated, hemorrhoids, polypi, etc., removed. Cleanliness after defecation should be observed. The prolapse should be replaced with the patient preferably in the knee-elbow position. The mass should be thoroughly lubricated with vaselin and gently pressed into the rectum. If a considerable portion is down, a large flexible bougie can be cautiously passed into the bowel, pushing before it the upper part of the descended gut, and pressure (taxis) should be employed evenly on the other surrounding portions with the fingers.

When the prolapse occurs frequently, a rectal supporter should be worn, such as a soft-rubber ball attached to the anus by means of a belt and T-bandage. A supporter can be improvised by employing a small roll of gauze covered with oiled silk and attaching it to a home-made T-bandage.

With children palliative treatment is more successful. The cause of the difficulty should be investigated and removed if possible, such as a polypus. The general health should be built up, mild laxatives should be administered, and the diet regulated to secure easy bowel action if there is constipation. If diarrhea is a factor, it should receive treatment. The child should be instructed not to strain at stool, and in many cases it is wise to have him defecate in the *knee-elbow* posture over a vessel to prevent prolapse as much as possible, or in the Sims posture, even at the expense of soiling the bedding or clothing, which can be easily changed.

If prolapse occurs, it should be washed with cold water or weak boric acid solution or an astringent alum solution, ʒj (4.0) alum to ℥viij (250 cc.) water, smeared with vaselin, and gently returned into the anus. After this the patient should remain in the recumbent position for about half an hour, preferably lying on the abdomen.

Radical measures must frequently be adopted. Cauterization of the prolapsed portion with fuming nitric acid or acid nitrate of mercury, as suggested by Allingham, or (preferably) with the thermocautery under anesthesia, has benefited or even cured cases. After cauterization the prolapsed part should be lubricated and returned into the anus. Strictures are occasionally produced by this procedure.

Other methods have proved successful, such as excision of elliptic portions of the mucous membrane with subsequent suture; extirpation of the prolapsed portion; revision or narrowing of the caliber of the rectum; suspension of the prolapsed rectum by attaching its mesentery to the abdominal wall; or suspension of the lower end of the sigmoid flexure.

Fissure of the Anus.—This condition quite frequently occurs with hemorrhoids. The fissure consists of an oblong tear of the mucous membrane of the anus, which gives rise to pain and spasmodic contraction of the sphincters. Some fissures are slight abrasions or they may be fairly large and deep. The edges may appear healthy, or be inflamed or indurated. Those of longer duration present the appearance of an ulcer. In fact, they are classified as irritable ulcer.

Anal fissure is usually caused by an injury or tearing of the mucosa at the edge of the anus. Excessive straining or the passage of dry hard scybalæ are factors. It is more common among women. The fissure may occur in any location, though most frequently at the posterior portion of the anus. It generally lies parallel and close to the external sphincter, though it may be higher up near the internal sphincter or above it.

Symptoms.—Severe pain in the rectum during defecation and at times tenesmus, persisting for a time thereafter, are present. The pain may be very severe in character, so that the patient dreads to have a stool, and increased constipation results.

A small fissure over the external sphincter usually causes greater disturbance than a larger one higher up. There may be reflex irritation of the bladder and a discharge of pus and blood from the rectum. From the pain and suffering the nervous system may become affected.

Diagnosis.—This is made from the symptoms and by local examination. The patient should lie on the left side and be told to bear down. On opening the anus with the finger and thumb, a fissure (crack) or small club-shaped ulcer can frequently be seen. It may be red and inflamed or, if more chronic, of a gray color with indurated edges. The pain may be so severe that before local examination it may be necessary to introduce a suppository of cocain, gr. ¼ (0.016). Rarely an anesthetic may be required.

For a fissure higher up, examination with the speculum may be necessary.

Treatment.—Hemorrhoids or polypi, if present, should be removed. The dilatation of the sphincter performed during this operation and removal of the cause of the fissure will usually cure the case. An incision through the base of the ulcer can be made at the same operation as a precaution.

Recent fissures are at times cured without operation. The patient should be in the recumbent position, hot lead-and-opium lotion can be applied externally to the anus if much pain, as heat relieves pain and spasm.

The following ointment and suppository are of value for the same purpose:

℞.	Ext. conii	ʒij	8
	Olei ricini	ʒiij	12
	Ung. lanol	q. s. ʒij	60.—M.

Ft. unguentum.

℞.	Ext. belladonnæ	gr. ⅓	02
	Ext. opii	gr. ½	03
	Ol. theob	gr. xv	1.—M.

One suppository.

Locally, the fissure can be touched with silver nitrate solution (I have used the pure silver nitrate stick twice a week), varying in strength from gr. 10 to 30 (0.6–2.0) to the ounce (30.0) of water every two to three days; on the alternating days a 5 per cent. cocain solution can be carefully applied, only a few drops; or the following ointment can be alternated with the silver application, or substituted for a time:

℞.	Ichthyol	gr. xxx	2	
	Lanolin	ʒij	8	
	Petrolati	q. s. ℥j	30	.—M.

Ft. unguentum.

or

℞.	Hydrarg. chlo. mit	gr. xv	1	
	Pulv. opii Ext. belladonnæ	āā gr. v	3	
	Petrolati	℥ss	15	.—M.

Ft. unguentum.

Gradual dilatation of the sphincter without anesthesia, as described under Hemorrhoids, especially with Roberts' dilator, is sometimes employed, but this procedure is painful.

Dilatation of the sphincter under anesthesia is *often found curative of obstinate cases.* Nitrous oxid is a valuable anesthetic for this purpose.

Some recommend infiltration of the fissure with a hypodermic of 1 per cent. cocain, and a free incision through its base to the sphincters.

An incision of the fissure under anesthesia, combined with sphincter dilatation, may be required.

Proctitis.—If proctitis is present with hemorrhoids, it must receive appropriate treatment, as described under the section on that subject.

CHAPTER XXIX

APPENDICITIS

(*Synonyms.*—Inflammation of the Vermiform Appendix; Perityphlitis; Appendicular Inflammation; Scolecoiditis.)

INFLAMMATORY conditions involving the right iliac region have been called by a variety of names: Iliac phlegmon; typhlitis (inflammation of the cecum); perityphlitis (inflammation of the covering of the cecum); and paratyphlitis (inflammation of the retroperitoneal tissue behind the cecum). Though typhlitis was considered to be the cause of most of the inflammations in the right iliac fossa, modern investigation has demonstrated that appendicitis is the chief factor.

Some go so far as to state that even stercoral typhlitis (an inflammation due to fecal accumulation) *does not exist.* This is an error, as C. A. McWilliams,[1] though admitting it to be rare, shows that an acute or chronic primary typhlitis may occur independently of appendicitis, dysentery, tuberculosis, actinomycosis, or cancer; and that it may be either idiopathic in origin or due to coprostasis.

Autopsy reports and findings at operations have confirmed this opinion. Cecal disease may go on to ulceration and perforation, with the formation of perityphlitic abscess or general peritonitis, while the appendix remains normal. Howard Kelly has reported 14 cases presenting primary lesions in the cecum, the *appendix being normal.*

Over 90 per cent. of inflammations are due to the appendix; the rest to the cecum.

The symptoms of typhlitis are usually indistinguishable from those of appendicitis and the indications for operation are the same. The literature on appendicitis is enormous, and I shall only mention the names of a few of those specially identified with the operative technic and investigation of appendicitis, namely: Reginald Fitz, Richardson, Sands, McBurney, Bryant, Bull, Weir, Fowler, Hartley, Dawbarn, R. T. Morris, Wyeth, Blake, Brewer, Hotchkiss, Deaver, and Howard Kelly.

Position of the Appendix.—It takes its origin generally from the posterior and median surface of the cecum, corresponding to McBurney's point (1½ inches from the anterosuperior spine of the ileum), on a line drawn from the spine to the umbilicus. The average length is from 3 to 5 inches, but it may vary markedly. The statistics as to its direction are quite variable. It may point downward and inward, downward behind the cecum, or upward, upward and inward, transversely inward, or outward. In many cases the appendix is quite long, and the position and length thus explain the variable locations of adhesions or abscess. It may, therefore, come in contact with the male bladder or rectum, with the uterus or right tube and ovary, or even the left tube, or with the small intestine, or it may pass up as high as the liver or right kidney, even to the left rectus, and close to the spleen. It has been found behind the peritoneum with no peritoneal covering. Bryant has reported it outside the peritoneal cavity in 3 cases. It has been found quite frequently in hernial sacs, and on several occasions in the scrotum. It quite frequently hangs free in the abdominal cavity.

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[1] Annals of Surgery, June, 1907.

Peculiarities of the Appendix.—The lumen of the canal is extremely narrow; the organ is bottle shaped, the narrowest part being near the entrance into the cecum. Gerlach's valve, a reduplication of the mucous membrane of the appendix near its origin from the cecum, makes the entrance of material more difficult, and also its exit. The circular muscular fibers are somewhat scanty. These factors tend to produce stagnation and predispose to infection.

There are many lymph-follicles (adenoid tissue) in the appendix, which usually persist up to the age of about thirty and then begin to retrograde, so that infection is easy, as in the tonsils. In many cases the appendix is completely surrounded with peritoneum, in some it is only partially covered, and the uncovered portion is in direct contact with the retroperitoneal connective tissue, which readily accounts for the so-called perityphlitic abscess. The appendix has a mesentery of its own, as a rule, the meso-appendix, but it is not constantly present. It rarely reaches up to the tip, but usually only one-half to two-thirds of the distance. It is believed to have some influence on the shape of the appendix, as when it is relatively short the latter may be bent. Crile,[1] in an analysis of 1000 cases of appendicitis, found that in a large percentage there was a short meso-appendix, causing fixation of the proximal part and leaving the distal end free, which tended to cause the appendix to fold on itself and interfered with the circulation (an anatomic angulation).

The blood-supply of the appendix is quite scanty. The vessels (the appendicular and a few cecal branches) Fowler believes to be functionally nearly end arteries, the most abundant being from the vessels (the appendicular) in the meso-appendix, so that the vascular supply of the tip of the organ is poor.

In females, Clado[2] holds that there is a third source of blood-supply from a vessel passing through the appendiculo-ovarian ligament (a fold of peritoneum passing from the meso-appendix to the broad ligament), and this possibly explains why appendicitis is less common in women, on account of the superior vascular supply.

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[1] Ohio State Medical Journal, June, 1907.

[2] Compt. Rend. Soc. Biol., 1897, vol. iv, p. 133.

Fowler holds that the blood-vessels may be primarily affected. Misplacement and malformation of the appendix may also have an influence. The peculiar anatomic conformation of the appendix may predispose to infection.

Etiology.—The chief cause of appendicitis is bacterial invasion, the most common of which present is the Bacterium coli commune. As a rule, the infection is a mixed one, streptococci being frequently associated. The proteus vulgaris has been present.

The Diplococcus pneumoniæ, staphylococci, the anaërobes, and the influenza bacillus have been found. These are the most frequent varieties and are found both in the appendix, appendical abscess, and in the general peritoneum (fluid of the), if peritonitis is present. In isolated cases, appendicitis is believed by some to be a local expression of a general infection, as associated with scarlatina, measles, rotheln, small-pox, chicken-pox, parotitis, influenza, and acute articular rheumatism. It has accompanied suppurative tonsillitis.

Typhoid, dysentery, and, more rarely, tuberculosis may be causes. Actinomycosis has also been demonstrated to have produced it.

Traumatism[1] or injury from lifting have been given as causes, but probably, if appendicitis occurs as a sequel, the organ was previously diseased or damaged, or the symptoms might be due to a circumscribed traumatic peritonitis.

Constipation as a factor in the production of appendicitis is a question of dispute. Fitz and Fowler believed that in the majority of patients the bowels acted regularly previous to the attack, and Riegel holds that constipation and diarrhea have no bearing on the subject. Though unquestionably many patients suffering from constipation never suffer from appendicitis, there is one type of case in which it may be a factor, namely, patients in whom there is a tendency to fecal accumulation in the caput coli and lower ileum, with resulting appendix symptoms, probably from circulatory interference, pressure, blocking of the appendix opening, and catarrh secondary to a slight catarrh of the cecum. I have seen several such cases in which there were the typic symptoms, which rapidly subsided after thorough bowel irrigation, ice-bag, and later cathartics and subsequent treatment of the constipation, with no subsequent attacks during eight or ten years' observation. To this same class belong the rare type of typhlitis due to stercoral ulcer to which McWilliams refers.

Dietetic indiscretions, in so far as they are productive of intestinal putrefaction or fermentation with increased bacterial activity, might be a factor in producing an acute attack in an appendix already damaged. Intestinal catarrh, involving the caput coli, in my own experience has been the direct cause of the catarrhal type of appendicitis.

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[1] Deaver, New York Medical Journal, June 15, 1907.

Entozoa, such as the trichocephalus dispar, oxyuris vermicularis, ascaris lumbricoides, tapeworm, and bilharzia, have been factors in the production of appendicitis. Movable kidney, through pressure on the mesenteric vein, has been given as a frequent cause by Edebohls. This condition is associated with splanchnoptosis, and the circulatory changes from malposition of the viscera are more probably factors. These conditions occur most frequently with women, yet appendicitis is less frequent in the female.

Foreign bodies entering the appendix, such as *grape seeds*, *cherry stones*, *pits*, *pins*, *buttons*, *gall-stones*, etc., are rare causes of appendicitis.

Fecal concretions are found frequently and have been mistaken for foreign bodies. Probably normally soft fecal matter enters and is expelled from the appendix. The fecal concretions are generally hard in character from absorption of water and are thickened by mucus. C. B. Lockwood has demonstrated that in many cases they consist of an inspissated mass of bacteria. It is easy to understand how such concretions, if of large size, can exert pressure and even be productive of ulceration, especially if there be any abnormal condition in the appendix. They have been found in one-third to nearly one-half of the cases. Small concretions might remain in an appendix without harm. They generally lie near the end (tip).

Right tubo-ovarian inflammation may be a factor in the production of appendicitis.

Age.—Appendicitis seems most frequent between ten and thirty years, Fitz stating that 50 per cent. occur before the twentieth year, and Einhorn 60 per cent. between sixteen and thirty years. It has been reported as early as the seventh week, but rarely before the third year.

Sex.—Men suffer from appendicitis much more frequently than women (from 2 to 3 or more to 1).

Varieties.—I will describe both the pathologic and clinical types of appendicitis.

From a pathologic standpoint, the following seems the best classification:

1. *Acute catarrhal appendicitis*, in which the mucous membrane is involved, being swollen and edematous, the submucosa injected with excessive secretion of mucus or mucopus.

This type is mild, and the appendix drains into the cecum with perfect recovery (endo-appendicitis, Fowler).

2. *Acute Diffuse Appendicitis.*—There is inflammation of the mucosa and thickening of the entire organ, which is rigid, tense, and infiltrated. By some it is classified as catarrhal, but this is incorrect.

The peritoneal surface is hyperemic. There may be erosions or small ulcers and a fecal concretion. There may be mucus or mucopurulent material in the lumen, or in some cases it may be narrowed or obliterated.

This type sometimes hangs free in the abdomen, but more generally is adherent to the adjacent peritoneal structures, and is characterized by the surgeons clinically as *acute non-suppurative appendicitis*. It may resolve without operation.

On the other hand, this pathologic type may be productive of abscess or perforation, when it would be placed under a different class by the surgeons.

3. *Purulent or Suppurative Appendicitis*.—This is a more advanced stage than the former. There is a definite pus-sac formed by the appendix. This type may also perforate and cause local abscess or general peritonitis.

4. *Gangrenous Appendicitis*.—This is characterized by necrosis, local or general. The tip is most frequently involved or the entire organ.

5. *Chronic Appendicitis*.—This may follow the acute, or the process may be slow and gradual from its incipiency. In some cases the changes have been found to be very slight, merely the evidence of a chronic catarrh of the mucosa, probably an extension from a chronic catarrh of the cecum. At times stenosis is present.

On the other hand, the organ has been found firm, slightly enlarged and thickened, the mucosa thickened, and the lumen narrowed. In some cases there may be a stenosis, with formation of a cyst. In others there are concretions or erosions, or partial obliteration of the lumen; or the appendix may become converted into a cord-like structure, embedded in a mass of thick peritoneal adhesions.

6. *Obliterative Appendicitis*.—A gradual involution process occurs in many individuals. The tube is thickened, the peritoneal surface smooth; the distal portion of the lumen may be entirely obliterated, and the organ becomes sclerotic and shrunken. Ribbert found these changes in more than 50 per cent. of subjects over sixty years of age. Normal involution seems to present no symptoms.

From a clinical standpoint the physician will find several types of acute and chronic appendicitis, readily deduced from the pathologic classification.

1. *Acute Simple Catarrhal Appendicitis* (*Endo-appendicitis*).—A catarrh of the mucosa of the appendix. This is of mild type and is often secondary to a colitis or to fecal impaction in the cecum. Unless there is occlusion, the inflammatory products usually drain out (non-suppurative type, with complete cure). There may be recurrent attacks.

2. *Acute Non-suppurative Appendicitis* (*Sometimes Incorrectly Characterized as Acute Catarrhal*).—The description is that of the Acute Diffuse Appendicitis, the entire organ being involved.

There is fibrinous exudation agglutinating the appendix to neighboring structures, and the meso-appendix is thickened and inflamed. There may be kinking or torsion of the organ, stricturing, or even obliteration, and fecal concretion may be left within it.

These cases often escape operation and may not have a recurrence, but there is great liability of the latter.

3. *Chronic Appendicitis.*—This may follow the acute, or the process be slow and gradual from the start. The chronic catarrhal cases may exhibit but little change, or the appendix may be thickened, the mucosa thick and hyperemic, and its lumen narrowed, obliterated, or strictured.

A fecal concretion may remain within it. It may present adhesions. It may follow an acute attack or be chronic from the onset.

These three types are characterized by *absence* of perforation, gangrene, or abscess formation; though type 2 may progress to type 4, with resulting abscess or perforation.

4. *Acute Suppurative Appendicitis (Formation of Abscess).*—Of this we have two clinical types, the pathologic *purulent appendicitis* (the appendix distended with pus), on the verge of perforation, or an inflamed appendix enclosed in an abscess-cavity containing a varying amount of foul pus. The walls of the cavity are formed by adjacent peritoneal surfaces, coils of intestines, cecum, and omentum bound together by adhesions.

The appendix lying in the abscess-cavity is congested and swollen, and may or may not be perforated or present areas of gangrene. It may be adjacent to the cecum or intestines or reach into the pelvis to the bladder or tubes and ovaries. Perforation, when present, is generally near the free end of the appendix.

5. *Gangrenous Appendicitis.*—The appendix is congested, swollen, thick, and red, with gangrenous areas of greenish-black color and usually already perforated. It is marked by the absence of *protective peritoneal* adhesions.

There may be a fatal septic peritonitis before perforation of the appendix. Patches of fresh fibrin may be present with serous or bloody turbid serum in the peritoneal cavity or adhesions with pus.

The condition is an acute gangrene due to thrombosis and occlusion of the blood-vessels. If the case is more chronic, some plastic peritonitis may be present.

A purulent appendix, an acute diffuse appendicitis, or a concretion may ulcerate through, so that any of these types—including the gangrenous—may be classified as *perforative appendicitis.*

6. *Harmful Involution of the Appendix* (Morris).—The vermiform appendix normally undergoes an involution process with replacement of the lymphoid, mucous, and submucous coat by connective tissue. R. T. Morris[1] notes that the nerve-filaments persist longer than other structures, and contraction of the connective tissue in some cases irritates these nerve-filaments, so that irritation of the ganglia of the bowel (Auerbach's and Meissner's plexuses) ensues and

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[1] Medical Record, April 6, 1907.

5. In the *septic gangrenous type* I have, in a fairly large percentage of cases, observed a toxemic type of diarrhea[1] with general abdominal pains at times, as the initial symptom, before localization of the appendix pain. This is evidently of septicemic character and has not to my knowledge heretofore been referred to as a symptom.

6. In some cases the thighs and knees are flexed.

Pain.—The pain may be sudden and violent, or at times intermittent and cramp-like, or even of a gnawing character or a dull ache. Sudden and violent pain in the initial stage does not by any means mean perforation, unless other symptoms are associated. In about one-half the cases the pain begins in the right iliac fossa; it may commence in the epigastrium, around the umbilicus, or even be diffuse, but gradually becomes localized within twelve to twenty-four or thirty-six hours, and usually sooner.

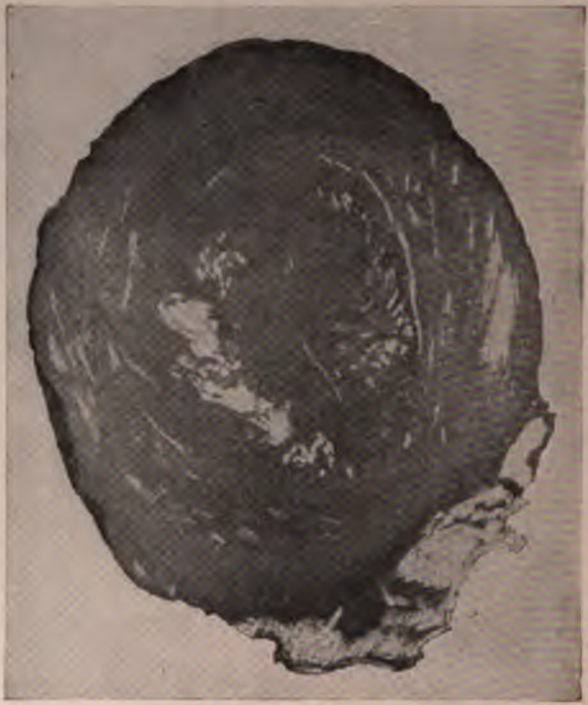

Fig. 219.—Transverse section of the appendix, showing replacement of the inner coats by connective tissue (Morris).

At times the pain is of a colicky type (the so-called appendicular colic, supposed by some to be due to constriction of the appendix in forcing out mucus through a lumen nearly occluded). Pain is increased on moving. It is often relieved by flexing the knees and thighs, especially the right thigh, and so relaxing the abdomen. This position is at times assumed by the patient.

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[1] Rudolph Schmidt assumes that the changes from normal in the intestinal flora of the feces, which occurs in appendicitis, may explain diarrhea in the early development of this disease; or possibly acute enteritis from dietary indiscretion may be the cause (Pain, by Rudolph Schmidt). The type of diarrhea with gangrenous appendix seems to the author ***peculiarly septic*** and is relieved by *appendectomy*.

Palpation of the Appendix.—Though some surgeons believe this to be an important procedure to render the diagnosis certain, Treves and Lockwood are very skeptical regarding the possibility of mapping out this organ. In the chronic cases palpation is of value, and the position and condition of the appendix can often be determined thereby.

In acute appendicitis the methods of forcible palpation recommended often necessitate the use of considerable pressure, and I believe the procedure highly dangerous. In the initial stages of acute appendicitis it is often impossible to at first determine the character of the attack, and traumatism in some cases can precipitate a rupture.

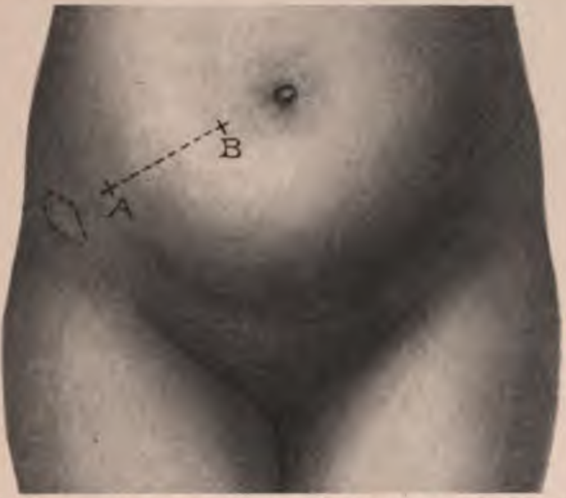

Fig. 220.—*A*, McBurney's point, and *B*, R. T. Morris' point (lumbar ganglia) in appendicitis.

In the acute cases gentle palpation[1] only should be used.

Muscular Rigidity.—There is usually rigidity of the right rectus muscle of varying intensity.

Tenderness on Pressure.—This occurs at McBurney's point and is of varying intensity, also at Morris' point, and Blumberg describes a new symptom. There are two points of great diagnostic value in appendicitis: tenderness at McBurney's point and at Morris' point (over the right lumbar ganglia).

McBurney's Point.—If a line be drawn from the anterior superior spine of the right ileum to the umbilicus, a point 1½ inches from the spine along this is known as McBurney's point; and deep-seated tenderness on pressure over this point is diagnostic of appendical inflammation when taken in consideration with other symptoms (Fig. 220). Deep pressure also often causes reflex epigastric pain.

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[1] Percussion, according to Rudolph Schmidt, will often demarcate the area of pain better than will palpation.

Mere superficial tenderness means irritation of the sensory nerves of the abdominal wall due to hysteria, etc.

Munro's point is slightly further out, where the same line crosses the outer border of the rectus.

Morris' Point.—"Take another point on this same line, but 1½ inches from the navel,[1] which lies over the right lumbar ganglia of the sympathetic system, and we have another point of diagnostic value when tenderness on pressure is located in this region.

"1. In the early *stages of an acute infective* process of the appendix, the right lumbar *ganglia are not tender*. (The left lumbar ganglia may be described for diagnostic purposes as lying 1½ inches to the left of the navel.) Under these circumstances the point here described is of *secondary importance*, while McBurney's point is of *prime consequence*.

"2. *A*. When an acute inflammatory process of the appendix has subsided, leaving a mucous inclusion or scar tissue, there may be no tenderness on pressure at McBurney's point, but there is tenderness *at the point here described* and no tenderness at the point of the left lumbar ganglia.

"*B*. When the appendix is undergoing an involution process, with replacement of its lymphoid *coats by connective tissue*, digestive disturbances and various local neuralgias may be due to irritation of nerve-filaments entrapped in the new connective tissue. There may be no tenderness at McBurney's point, but there is persistent tenderness at the point here described. There is no tenderness at the point of the left lumbar ganglia."

The above condition constitutes fibroid degeneration of the appendix.

"*C*. When the appendix is congested without the presence of infection, as in many cases of loose kidney, there may be little or no tenderness at the point here described. There is no tenderness at the point of the left lumbar ganglia."

The author wishes to state, as before, that this type of appendical congestion he believes *not due to loose kidney*, but dependent on the *enteroptosis*.

"In irritations of pelvic origin, both right and left lumbar ganglia are tender. Take, for illustration, a case in which the appendix and the right Fallopian tube are bound together by adhesions. We are to decide whether certain symptoms proceed from the appendix or from the Fallopian tube. If the symptoms proceed from the appendix, the point here described is tender alone. If the symptoms proceed from the Fallopian tube, *both right and left lumbar ganglia* are tender together.

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[1] Surgical Section, New York Academy of Medicine, Dec. 5, 1907. The author here quotes Morris' deductions.

"To recapitulate: A patient comes in with the appendix in the form of a question mark. Right lumbar ganglia tender alone —appendix trouble. Right and left lumbar ganglia tender together —pelvic trouble. Neither right nor left lumbar ganglia tender— trouble somewhere cephalad from pelvis and appendix."

Blumberg[1] describes a sign pointing to peritoneal irritation or inflammation, which he considers will be found of assistance in the diagnosis of all peritoneal conditions, and especially of appendicitis. It consists in the fact that in palpating the abdomen in the neighborhood of an area of inflamed peritoneum, not only is the downward pressure painful, but if the examining hand is suddenly removed, the *abrupt recoil* of the abdominal wall also gives rise to pain. He has found that during an acute attack of appendicitis with peritoneal involvement patients invariably state that the pain caused by the sudden removal of the hand is greater than that caused by the pressure, while if the inflammatory process is subsiding, the two painful sensations first become equal in intensity, and finally the pressure pain is greater than the other. He further believes that the sign is especially valuable in determining whether or not operation is indicated in early cases, since its presence indicates that the peritoneum has already begun to take part in the inflammation. Its sudden appearance is, therefore, a danger signal, while its gradual diminution in intensity points to a subsidence of the peritoneal reaction. The advantage of the method is that it does not require an absolute estimation of the degree of pain caused by the palpation, which is often difficult to obtain, but demands simply a comparison of the intensity of the two painful stimuli which most patients are able to determine accurately.

S. J. Meltzer's Method.—Meltzer extends the right knee, at the same time flexing the right thigh, while making pressure over McBurney's point. This projects the psoas muscle against the appendix and causes deep-seated tenderness in the appendix to be more readily appreciated.

H. Illoway[2] holds that forced flexion and especially forced extension of the right thigh will cause pain in the appendix if inflammation is present.

In many cases, especially in the acute catarrhal conditions or in the most virulent gangrenous type, no *induration* or swelling can be appreciated on palpation. As I have stated, forcible palpation in the endeavor to appreciate the appendix should be avoided.

Percussion.—In cases with adhesions, exudation without pus, or of abscess, a boggy or, rarely, fluctuating mass can be appreciated in the right iliac fossa, and percussion will give a dull area in the region of the cecum. Fecal accumulation as a result of constipation must be differentiated by the methods I have already indicated.

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[1] Münchener med. Wochenschr., June 11, 1907.

[2] Archives of Diagnosis, July, 1908.

At times great irritability of the bladder is associated with this condition, and the urine may be scanty and contain indican and albumin, and even acute nephritis may be present.

Rectal and *vaginal examination* may sometimes aid in localizing the condition if the appendix or abscess lie in the pelvis. .Inspection may occasionally show protrusion on the right side or the distention of general peritonitis.

If *tumor is present,* it may be variable in size, more frequently in the right iliac fossa. Its position, however, depends on that of the appendix, as described on page 603.

Temperature.—An initial chill is rare. In the acute cases fever, even though slight, is present in the early stages. It may be only 99.5° F. or keep low, or even rise to 101° or 102° F., or to a considerable height.

Sometimes with circumscribed abscess there may for a time be only moderate temperature, and some cases of the virulent type will suddenly perforate, though the temperature be not high. In both of these, physical examination, the blood-count, and general symptoms will aid the diagnosis.

In general, a rise of temperature is significant of an active process, even though the temperature increase may be slight in degree and gradual in character. A slight increase in rapidity of the pulse is also suggestive of an acute process, and at times this is noticeably *much out of proportion to the temperature,* especially the *rapid pulse* in *gangrene* or *sudden perforation.*

Gastro-intestinal Symptoms.—Loss of appetite and coated tongue are present. Emaciation may occur in cases of long duration.

In the severer cases vomiting is quite common. It may be one of the first symptoms occurring with the pain, and then cease, or it may continue for several days. On the other hand, it may come on later in the attack and denote an exacerbation of the inflammation. It consists of the stomach contents, mucus, and bile; and in some cases it may be feculent. Associated with it there is at times hiccough.

Black vomit ("vomito negro appendiculaire") is the result of toxemia, producing hemorrhagic necrosis of the mucosa of the stomach and hematemesis.

A few cases have also been reported of intestinal hemorrhage associated with jaundice and albuminuria. These conditions are all evidences of a fatal sepsis. Acute ectasia or acute gastro-intestinal dilatation may also occur as complications.

Bowels.—In some cases the bowels are regular until the attack, when constipation ensues. In others there may be a previous history of constipation. Diarrhea of a toxemic type may be one of the initial symptoms in acute gangrenous appendicitis. It is probably of septicemic character. I have noted it in several such cases. Intestinal paresis may occur as a complication.

Tumefaction or Abscess.—In cases of acute appendicitis in which adhesions are present, a tumor due to adhesions and exudation, or an actual abscess, the position of the mass is dependent upon the position of the appendix primarily and then upon the direction of the extension of the inflammation or burrowing of the pus. It may pass down into the pelvis and produce bladder and rectal symptoms, or those pointing to the tubes and ovary (right), and be palpable through the rectum and vagina. It may point below Poupart's ligament or simulate a psoas abscess. It may pass around in front of the cecum and superficial edema be noted in this region, or it may pass posteriorly to the cecum and cause pain in the flank or back, with swelling, and produce perinephritic abscess or lumbar abscess. It may pass inward to the left and produce obstruction through pressure or bands. It may pass upward and produce subphrenic abscess and even perforate the diaphragm, pleura, and lungs. It may develop in a hernial sac.

At times the tumor appears at the lower border of the liver, when the tip of the appendix lies in this region. The condition must then be differentiated between retroverted appendical abscess and gall-bladder or renal inflammation, such as infarction.

Brewer has shown that *tenderness at the costovertebral angle* is diagnostic of the latter; the urine analysis is also important, and especially differential analysis after catheterization of both ureters. The history will usually point to appendicular inflammation or disease of the gall-bladder, but operative procedure will alone settle some cases.

The value of Head's zones of cutaneous hyperalgesia as an aid to differential diagnosis will be referred to under that section.

The elements in the diagnosis of abscess formation are the gradual increase of the local tumor and the aggravation of the general symptoms. The abscess may perforate and cause a general peritonitis or the inflammation may extend to the peritoneum without perforation, so that there may be a slower process with various sacculated collections of pus.

The abscess may rupture through the skin or empty into the cecum, colon, small intestine, bladder, rectum, or pelvis of the kidney.

Fulminating Type of Acute Appendicitis.—This is the most dangerous and fatal of all. This type of appendicitis can be subdivided into two clinical varieties. Both are characterized by the rapidity of the pathologic changes in the appendix and by the absence of protective peritoneal adhesions, so that general peritoneal infection occurs quite rapidly.

In the first class the pain in the region of the appendix and some of the subjective and objective symptoms are acute and quite marked, but not all of them.

In the second class, and by far the most dangerous because frequently undiagnosed until the general infection has occurred, the patient complains of no marked subjective symptoms; in fact, may say he is quite comfortable, and the objective symptoms are not marked. There are peculiarities in the pulse, temperature, and especially in the blood changes which tell the story.

Acute gangrene of the appendix, with or without perforation, is the pathologic condition generally found on operation; though I have also seen an acute diffuse or purulent inflammation of the appendix, either with perforation or without it, produce the condition. In some cases perforation was apparently due to ulceration from a fecal concretion which was found in the cavity. Perforation undoubtedly is present in many cases, but I agree with Riegel that acute virulent infection of the peritoneum, commencing in the region of the appendix, will produce the same clinical symptoms.

In the first class of cases the patient may have given a history of previous attacks or have had some indefinite abdominal symptoms for several days, or may be attacked without warning, as in the middle of the night, with severe pain in the abdomen. It may not at first be referred to the appendical region, may be epigastric or umbilical pain, but gradually localizes there. There is no tumor, the muscles are rigid, the appendix tender. The abdomen rapidly distends. The patient is anxious and looks sick. The temperature at first may not be elevated much. The pulse is *rapid and out of proportion* to the temperature. General symptoms of peritonitis rapidly ensue. In others the pulse may be slow at first and a low temperature with local signs of appendicitis, but acute history, nausea, vomiting, and marked constipation. The pulse later becomes more rapid, high temperature, distention, coated tongue, and general tenderness.

In others the first symptoms are of collapse, with subnormal temperature, rapid pulse, cold and clammy skin, respiration increased in frequency, followed by symptoms of general peritonitis.

In the second class of cases the condition may be very deceptive. The patient, in perfect health, may suddenly complain of general abdominal pains. I have seen a number with *diarrhea* of toxic character as the initial symptom. The patient may impute the symptoms to dietary indiscretions. The tenderness may be diffuse or equal on both sides, and gradually it localizes in the appendix region. Even so, the tenderness at McBurney's point may not be very acute, and the rigidity of the right rectus not very marked or very slight. The patient may state that he feels quite comfortable. The temperature may be moderate (100° F.) and the pulse 100.

The temperature tends to gradually creep up, the pulse to increase in rapidity out of proportion to the temperature increase, which last may be slight, *and the character of the pulse changes;* the patient looks more sick, but still complains of no special symptoms except slight pain or exacerbations of it in the right side, and still feels comfortable.

The blood examination shows in many cases moderate leukocytosis (14,000 to 16,000), but marked increase in the polynuclears (88 to 92 per cent.). Hyperinosis is present. The patient presents as yet no symptoms of general peritonitis, but nevertheless the sepsis is marked in these cases, and unless immediate operation is performed will have a virulent type of septic peritonitis.

A patient of mine recently operated on by Hartley presented the mild type of symptoms just described: Diarrhea at 4 P. M.; no appendical pain or tenderness; localized appendical pain at midnight, when the surgeon was at once called in. Differential blood-count at 8 A. M. and 1 P. M., showing a gradual increase in polynuclears and low leukocytosis (15,000 to 16,000). The patient was quite comfortable; no distention; slight pain over appendix, but the temperature and pulse slowly creeping up.

At 5 P. M. operation at the New York Hospital; acute gangrene of appendix and commencing peritonitis; ultimate recovery.

The examination of the blood is thus imperative when possible.

The Blood in Acute Appendicitis.—Hyperinosis (increased fibrin in the blood) has been demonstrated by E. E. Smith and Bartlett[1] to be more marked in direct proportion to the involvement of the serous surface, and hence, *depending on its degree*, is suggestive of proportional peritonitic infection.

One of the *most important factors in the determination as to operative procedure in appendicitis and as to the relative severity of the case is the differential leukocyte count.* Charles Langdon Gibson has especially pointed out that it is the disproportion between the percentage of polynuclear cells and the total leukocytosis that is important.

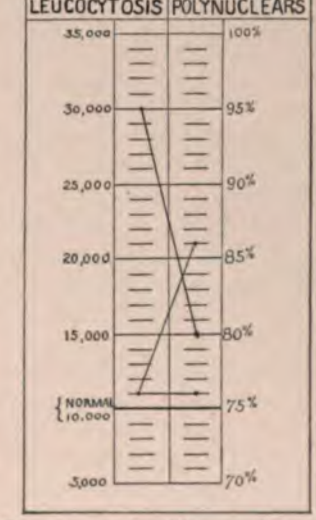

Fig. 221.—C. L. Gibson's differential chart for leukocytosis.

The chart (Fig. 221) assumes that 10,000 leukocytes per cubic millimeter is the upper limit of ordinary normal leukocytosis, and that 75 is the normal percentage of polynuclears. Gibson further assumed that in inflammations which are well resisted the polynuclear cells are increased approximately by 1 per cent. for every 1000 leukocytes above the normal 10,000 per cubic millimeter. Then in the chart the *horizontal* line will indicate a leukocyte count of 11,000 with 76 per cent. of polynuclears, whereas the *rising* line represents a leukocytosis of 11,000, but with 86 per cent. of polynuclears.

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[1] Blood Reactions of Inflammation, Med. Record, Feb. 8, 1908.

If the line connecting the total leukocytes and the percentage of polynuclears runs fairly *horizontal*, it indicates a lesion that, whether severe or not, *is well borne* and, therefore, of good prognosis.

If the line runs *upward* from the leukocyte to the polynuclear side, it indicates a rather severe lesion and less resistance.

Fatal cases all have a rising line.

A *falling* line (*e. g.*, leukocytosis of 30,000 with 80 per cent. of polynuclears) means a *mild lesion;* in appendicitis it would probably indicate an abscess well shut off, with little febrile or constitutional disturbance.

Gibson's conclusions are as follows: The differential blood-count and its relation to the total leukocytosis is the most valuable diagnostic and prognostic aid in acute surgical diseases that is furnished by any of the methods of blood examination.

It is of chief value in indicating fairly consistently the existence of suppuration or gangrene, as evidenced by an increase of the polynuclear cells disproportionately high as compared to the total leukocytosis.

E. E. Smith[1] shows that it is an indicator of the *activity of the process* and not *invariably of gangrene;* but if the absolute *leukocytosis is low* (below 15,000), with *high polynuclears*, it is *probably* gangrene.

Gibson further holds that the greater the disproportion the surer are the findings, and in extreme disproportions the method has proved itself practically infallible.

As the relative disproportion between the leukocytosis and the percentage of the polynuclear cells is of so much more value than the findings based on a leukocyte count alone, this latter method should be abandoned in favor of the newer and more reliable procedure.

The negative findings, showing no relative increase or even an actual decrease of the proportion of the polynuclears cells, while of less value, show with rare exceptions the absence of the severer forms of inflammation.

In its practical applications the method is of more frequent value in the interpretation of the severity of the lesions of appendicitis and their sequelæ.

Though N. E. Ditman, in a paper read before the Obstetrical Section of the Academy, November, 1906, criticizes the differential count as of doubtful value, Sondern[2] shows that in cases quoted the pus was encapsulated in such a way that no toxic absorption occurred, and also demonstrates that polynuclear increase may occur in other than suppurative conditions.

It is a well-known fact that such encapsulated abscesses may occur without marked constitutional symptoms, *but physical signs determine* their presence.

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[1] Blood Reactions of Inflammation, Medical Record, Feb. 8, 1908.

[2] New York Medical Journal, June 26, 1907.

In this type the processes are less active, and this is probably the chief cause.

I have seen a very low leukocytosis with a general fatal peritonitis, the system evidently being overwhelmed by the poison. The physical signs, with increased *polynuclears* and *hyperinosis*, aid our diagnosis in exceptional cases.

A low leukocytosis with high polynuclear count also shows poor resisting power to the infection.

To recapitulate: We may have numerous types of acute appendicitis, which clinically are as follows:

1. Simple catarrhal appendicitis with mild symptoms, lasting a few days to a week or ten days; often secondary to colitis, intestinal disturbances, or fecal impaction in the caput coli; blood changes are moderate and the attack soon subsides under medical treatment combined with the ice-bag. It may never recur.

2. Acute appendicitis (diffuse), symptoms more severe; may be adhesions or slight exudation; temperature higher; more marked tenderness; tumor often palpable, but may disappear. It may become chronic or recurrent.

3. Acute appendicitis, with abscess, chills, tumor, etc., present. Abscess may perforate.

4. Fulminating type, perforation or gangrene.

Remote Effects of Acute Appendicitis.—They are as follows: Hemorrhage from perforation of a blood-vessel; suppurative pylephlebitis; thrombosis of the iliac or femoral veins; pulmonary embolism; strangulation of the bowel; subsequent symptoms may occur due to incomplete removal or subsequent adhesions. Hartley has noted intestinal paresis in mild cases, with symptoms of ileus resulting; also infection of the mesenteric glands, subsiding in some cases after appendectomy, and in others causing subsequent inflammation.

Chronic Appendicitis.—This may follow an ordinary acute attack or be chronic from its incipiency.

The latter often is secondary to chronic intestinal catarrh. The patient has frequently a continuous feeling of discomfort in the appendical region; slight tenderness on pressure; generally intestinal and frequently nervous disturbances associated, also constipation or gastric disturbances. Slight tenderness or discomfort on pressure at McBurney's point. This may disappear for periods. At times no tenderness can be elicited at McBurney's point, but is noted at Morris' point. There may be exacerbations of acute attacks.

Indiscretions in diet often cause exacerbations of the symptoms. In some of these cases there is simply a chronic catarrhal condition, but more frequently angulation with adhesions.

This is the type where palpation by Edebohl's method is safe and of value, and where at times the enlarged appendix can be appreciated.

The patient lies on his back[1] with the thighs flexed, and the examiner, placing three or four fingers of the right hand, palm side downward, draws them over the abdomen from the umbilicus to the anterior superior spine of the ileum, exerting considerable pressure. The appendix can be recognized as a firm cord. Morris reinforces this hand with the three fingers of the left hand.

In the chronic cases there is generally no temperature unless subacute or acute exacerbations occur.

Harmful Involution of the Appendix.—Symptoms of harmful involution of the appendix (Morris):

1. Symptoms of auto-intoxication with attacks of headache, nervousness, poor appetite, etc. 2. Intestinal dyspepsia. 3. Discomfort in the appendical region. 4. An appendix feeling hard and narrow on palpation. 5. Hyperesthesia of the right lumbar plexus.

There is persistent distention of the cecum and ascending colon, with gas and a sensation of discomfort in the appendical region. The patient has a tendency to press upon the abdomen at that point or to lean against a table. The sensation may pass away for a few hours or days, but tends to recur and last for years.

On palpation the involution appendix feels narrow and hard. There is no history of acute or chronic appendicitis. The type generally occurs in those over twenty-five or thirty years of age.

Diagnosis and Differential Diagnosis.—Abdominal pain, becoming localized in the right iliac fossa; tenderness at McBurney's point; rigidity of the right rectus; temperature; rapid pulse; gastro-intestinal disturbances; in some cases the presence of tumefaction and in others the subsequent development of peritonitis—are all diagnostic of appendicitis.

In addition there are the differential leukocyte count and hyperinosis, which are an aid to diagnosis.

Renal colic with calculus impacted in the right ureter may simulate appendicitis, but there is the history of acute pain in the kidney, burning sensation of the urine, and drawing up of the right testicle, with sand, gravel, or blood, etc., in the urine.

With Dietls' crisis there is the movable kidney, the history of the attack, and the kidney is swollen and sensitive.

In intestinal colic the pain is relieved after passage of flatus.

In biliary colic the pain radiates to the back and up to the right shoulder, usually with a previous history of gall-stones, etc.

In perforation of the gall-bladder or duodenum the contents gravitate toward the appendix; and this possibility must always be considered in apparently acute perforative appendicitis. In these cases the sudden acute pain occurs in the epigastrium and right hypochondrium.

In women the differential diagnosis between a low appendix and salpangitis is sometimes difficult, as they frequently are associated.

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[1] It has been at times recommended to examine with the patient standing erect or bending slightly forward, but the dorsal posture is preferable.

With pneumonia, especially central near the right base, and with diaphragmatic pleurisy there is, occasionally in the early stages, pain *transferred to the right iliac fossa* and mistaken for appendicitis. The physical examination of the lungs and pulmonary symptoms should be carefully observed.[1] This possibility must be considered.

In typhoid fever there may be pain in the right iliac fossa and appendicitis is often a complication.

In simple typhoid there is no leukocytosis, but leukopenia.

In typhoid with appendicitis, in addition to the right iliac pain and tenderness, the presence of leukocytosis, increased polynuclears, and hyperinosis make the diagnosis of appendicitis.

As an aid to differential diagnosis in affections of the viscera, the determination of Head's zones (cutaneous hyperalgesia) is of value. This is especially true, I believe, in the differentiation of appendicitis from the conditions to which I have just referred. Head found that in many visceral affections, if the sensitiveness of the skin was tested by running a pin point over the cutaneous surface, there could be shown to exist areas over which there was a more or less hypersensitiveness to pain. These areas were constant and distinct, could be mapped out on the surface of the skin, and, when present, were almost an infallible sign of an affection of the organ to which they corresponded. The skin tenderness was superficial and extended over definite areas which never overlapped one another. Each area or zone of hyperalgesia had a "maximum region" which often corresponded to the seat of pain. These areas were sensitive to heat and cold, but not to simple touch.

The areas *corresponded to segments of the spinal cord,* not to the distribution of peripheral nerves or spinal nerve-roots. The zones were named according to the segments of the cord: cervical, 1 to 7; dorsal, 1 to 12; lumbar, 1 to 5; sacral, 1 to 4. They were broader in front at the median line, narrowed at the side of the body, and again broaden out near the spinal column. The zones, as a rule, never extend beyond the median line in front or behind. Head gives the following zones for the abdominal viscera:

Stomach, sixth, seventh, eighth, and ninth dorsal. Cardiac end, sixth and seventh dorsal, right. Pyloric end, eighth and ninth dorsal, left.
Liver, eighth, ninth, and tenth dorsal, right.
Gall-bladder, eighth and ninth dorsal, right.
Intestines, ninth, tenth, eleventh, and twelfth dorsal.
Colon, ninth, tenth, and eleventh dorsal.
Cecum and appendix vermiformis, tenth and eleventh dorsal, right.
Kidney, tenth dorsal, sometimes eleventh dorsal.

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[1] Determination of Head's zones is of value; the zone for the lungs is from first to the ninth dorsal segment, chiefly the third, fourth, and fifth, which give hyperalgesia over the thorax. With appendicitis hyperalgesia is below the umbilicus (page 614).

Ureter, eleventh and twelfth dorsal, first lumbar.

Bladder (first?), second, third, and fourth sacral.

Uterus, tenth, eleventh, and twelfth dorsal, first lumbar.

Appendages, eleventh and twelfth dorsal, first lumbar.

Head first tested sensitiveness to pain by pinching up folds of skin and later by stroking the skin with the point of a sharp pin. Elsberg[1] and Neuhof suggest the following method of examination:

"A sharp pin is held between the thumb and index-finger of the right hand, the nail of the index-finger resting on the patient's skin. The pin is then made to traverse slowly the surface of the skin, care being taken that the nail of the index-finger presses equally along the area examined. The patient is instructed to say 'now' as soon as the pin stroke becomes painful.

"In examining the skin of the abdomen for hyperalgesic areas, the pin traverses the abdomen from side to side and from above downward; the points at which the patient complains of pain are marked. In this manner it is possible to map out areas on the skin, and when such an area has been found, the pin is made to approach it from all sides, so that its form and position can be determined. Care must be taken that the pressure of the pin point remains constantly the same, especially as the pin passes over the groin and slips off the costal border or over the crest of the ileum.

"After the zone has been thus mapped out on the skin the procedure is repeated a second time, and now it is a good plan for the operator to control both patient and himself by keeping both the patient's and his own eyes away from the pin.

"The hyperalgesia is sometimes so marked that the patient will shrink or cry out as soon as the border of the zone is reached. In very young children the examination is useless, but older children will give correct answers.

"If the examination is carried out in the manner above described it will be possible in a large number of patients with visceral affections to map out areas of hyperalgesia extending from the median line in front to the spines behind. The 'maximum' areas can often be mapped out lying within the boundaries of the zones; sometimes only the 'maxima' are present. Sometimes several 'maxima' are found in one zone."

The zones appear early in the course of visceral affections, and frequently persist throughout. They have been reported as appearing very early; for example, in the commencement of acute appendicitis while the pain was still in the epigastric region and there was no local tenderness at McBurney's point, the zone for the appendix was discovered. Shortly after the typic symptoms appeared. One must remember the following (Elsberg):

1. The characteristic zone may appear after palpation of the diseased organ.

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[1] American Journal of the Medical Sciences, Nov., 1908.

2. The hyperalgesic zone will not appear on examination until fifteen to thirty minutes have elapsed after removal of the ice-bag or hot-water bag, if such have been applied.

3. The disappearance of the zone, as a rule, follows relief of the lesion of the affected viscus.

4. The zones may disappear temporarily after repeated examinations in close succession. Later they reappear.

5. The disappearance of the zone, together with persisting or increasing symptoms, is probably a sign of ill omen.

6. The zones are not invariably present. While the absence of a characteristic zone in a suspected affection of an abdominal organ *does not mean* that there may not be disease of that organ, the *presence* of the *zone* means that there *is an undoubted lesion.* From this one must *not* conclude that the viscus which gives the zone is the one which causes *all the symptoms,* for we may get a zone from an organ which is secondarily affected.

7. The presence of areas of skin hyperalgesia *corresponding to several* viscera may mean a *combined lesion of several* adjoining *viscera,* although it may occasionally *mean disease* of the *spinal cord itself.*

8. The presence of a Head zone alone must not be the only factor in arriving at a diagnosis, but it must be used in conjunction with other signs and symptoms. When one is in doubt as to which of several viscera is the seat of the lesion, the presence of the characteristic zone has been an aid; for example, in differential diagnosis between appendicitis (with retroverted appendix) and kidney and gall-bladder disease, or between appendicitis and salpingitis. It seems preferable to adopt Elsberg's method and speak of the zones by the names of the viscera to which they belong. The position of his zones vary a trifle from Head's, and are as follows:

Thus, the stomach zone corresponds to the seventh, eighth, and ninth segments of Head (according to Head, sixth, seventh, eighth, and ninth); the gall-bladder zone, to the eighth and ninth segments on the right side (same as Head's diagrams); the appendix zone, to the tenth and eleventh segments of Head on the right side. It will be found, in the description of the zones, that the limit of the posterior portions is not absolute. Thus, in describing the gastric zone, that it extends from the sixth to the tenth vertebræ approximately. The zones, except the gastric zone, stop sharply at the posterior median line, but their upper and lower margins are more variable.

The zone appears on that side of the body on which the affected organ has its nervous connections, the side on which the organ is normally situated. If an organ belongs on the left side, the hyperalgesic zone will be found on that side, even if the organ, through disease or mobility, lies on the other side of the body.

Those areas are called "objective zones" when the patient suffers actual pain as the stroking pin enters them. All less painful zones will be called "subjective zones." By an "anterior zone" we mean an anterior maximal area; by a "posterior zone," a posterior maximal area.

The Stomach.—The complete gastric zone was found to be uncommon. It extended as a broad belt all around the body. At times only a portion of it showed on examination.

In the median line in front it extends from the xiphoid almost to the navel; it then passes upward and backward on both sides toward the spine, where it extends from the sixth to the tenth vertebra (approximately). Incomplete zones are more frequent, either an anterior portion extending to the right or to the left, or on both sides of the anterior median line.

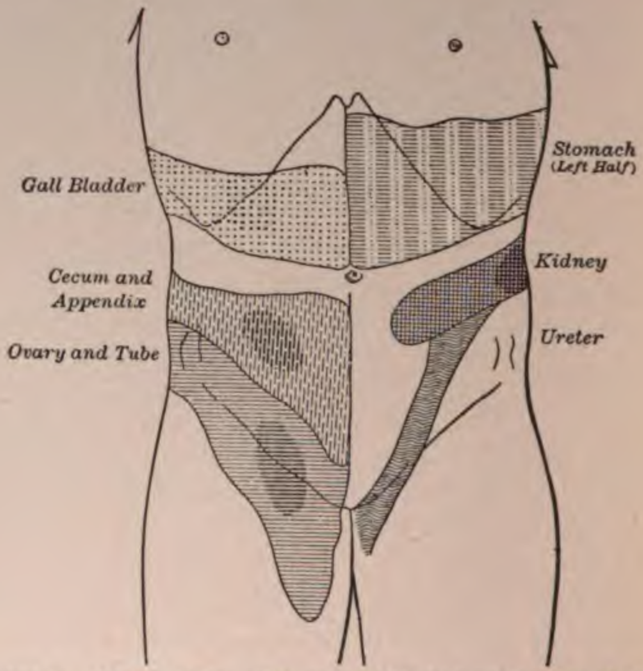

Fig. 222, *a*.—Head's zones. The general location and outline of the zones of cutaneous hyperalgesia for some of the abdominal viscera. Anterior view. The maxima are deeply shaded. Only the left half of the gastric zone is given. The ureteral zone consists of a series of maxima (diagrammatic) (Elsberg and Neuhof).

In Fig. 222, *a* and *b*, the various zones are depicted on the anterior and posterior surfaces of the body.

The Duodenum.—The duodenal zone lies between the gall-bladder and the appendix zones. It lies almost completely to the right, but occasionally extends slightly to the left of the anterior median line. Anteriorly it is broad; its upper limit is about on a

In some patients the presence of a zone of hyperalgesia has been the only localizing sign.

The gall-bladder zone lies in the right half of the abdomen, above the level of the umbilicus. The complete zone starts exactly at the median line in front, extending from some distance below the xiphoid to a short distance above the navel. Tracing it backward, it slants obliquely upward and becomes narrow, passing partly over and partly below the costal arch. It is narrowest at the midaxillary line, where it is about 2 inches wide. Posteriorly it becomes broader, and at the spines it is about as wide as in front. In some cases more or less of the anterior portion only has been present (maximal area).

Kidney and Ureter.—The kidney zone is wide at the posterior median line, where it begins, and gradually narrows anteriorly. Its greatest breadth is at the spinal column. It narrows to make a triangular area, with a rounded apex, situated a little to that side of the anterior median line on which the zone lies. It never quite reaches the anterior median line. Each zone is strictly limited to its half of the body. There is no difference in contour between the right and left kidney zones. The kidney zones are complicated by the additional ureteral zones that are present in certain cases. The ureteral zone springs, so to speak, from the lower margin of the kidney zone at the anterior axillary line. In an average adult it is about 3 inches wide at its beginning. It narrows in its downward course, and passing obliquely downward and forward, it terminates on its side of the penis and scrotum in the male; the labia in the female. After the first narrowing it widens again well below the umbilical level. In the male, it can be ascertained that the zone spreads fan shape to the anterior median line over the pubic area and its half of the scrotal and penile skin. There are anterior and posterior kidney maximal areas. The ureteral zone seems to be made up of a series of maxima. The kidney and ureteral zone is most often present, as in the other intra-abdominal affections, in the presence of pain and tenderness.

In the differential diagnosis of septic infarction of the kidney, pyelonephritis, etc., from retroverted appendix (appendicitis), determination of the kidney zone is of value, taken in connection with Brewer's sign, urinary analysis, etc. (author).

Vermiform Appendix.—The zone begins at the median line in front, sometimes a little to its left, from a point a short distance below the umbilicus to one equally distant from the symphysis pubis. It narrows toward the anterior axillary line to a width of about 2 inches (average adult). From this line it widens and spreads to the posterior median line from the eleventh dorsal to the second lumbar spines (approximately). At the anterior median line there is often a tongue-like downward extension of the zone (Fig. 222, *a* and *b*). There is an anterior maximal area which is sometimes present alone. It may be that the "appendix" zone is really an "appendix and cecum" zone, because the cecum is so frequently involved in appendicitis. Sometimes, when an ice-bag has been employed over the appendix region, only the posterior half of the zone is present.

Diagnosis has been aided in a considerable number of the patients by the presence of the zone, especially in that large class of acute cases in which the abdomen is rigid and there is no palpable mass. The zone has been of the greatest value in helping to differentiate between diseases of the appendix and those of the gall-bladder or right uterine adnexa.

The absence of a zone is of no significance. If a patient complains of symptoms which resemble appendicitis, and a zone is not present in the right lower abdomen, it is well to look elsewhere for hyperalgesia. Thus in cases of *beginning pneumonia* that had considerable *pain, tenderness,* and *rigidity in the right iliac region,* the presence of *hyperalgesia over the thorax first lead to careful examination of the lungs.*

Intestines.—Head gives the zone as corresponding to the ninth, tenth, eleventh, and twelfth dorsal segments of the spine. Elsberg does not find these uniform. He shows, however, uniform zones in ileocecal tuberculosis and in perforation of the ileum.

Ileocecal Tuberculosis.—There is a large area of hyperalgesia occupying the whole right lower abdomen down to Poupart's ligament, often extending a little to the left of the median line, and posteriorly becoming lost about the posterior axillary line.

Perforation of the Ileum.—The zone resembles that of ileocecal tuberculosis, but extends more to the left of the median line.

Uterus and Adnexa.—Head describes differences between the zones for the uterus, the ovary, and the tube. Elsberg differs somewhat: The zone for the right adnexa lies on the right half of the median line; that of the left adnexa on the left half; the zone for the uterus is a combination of the two. There is no difference between the zones for the right and left adnexa. Beginning some distance above Poupart's ligament, the upper margin of the zone runs parallel to it, and pursues this obliquely upward course to the spine of the second lumbar vertebra (approximately). The lower margin is a long, tongue-like process that extends half-way down the thigh on its inner aspect. The lower margin, as it passes a short distance below the anterior superior spine of the ileum, approaches the upper, the average breadth of the zone here being 3 inches. The lower border then passes horizontally backward over the buttock to reach the posterior median line partly over the sacrum. Sometimes the upper half of this zone is better developed, sometimes the lower; these may be considered maxima.

Diagnosis in the diseases of the uterus has not been aided by the presence of a zone. Elsberg states that in about half of the cases of dysmenorrhea and of endometritis with pain, the zone was present. Some of the cases of retroflexion, retroversion, anteflexion, and prolapse showed the zone. It was present in the 5 cases of uterine polyp that he observed (all of them had pain). It was not present in tumors of the uterus, except in a few cases.

In diseases of the tubes and ovaries, especially those of the right side, the zones have been of diagnostic value.

I can substantiate these observations of Elsberg and Neuhof, and believe that the tests[1] for Head's zones are a valuable aid in differential diagnosis, especially in appendicitis.

Prognosis.—There is always an element of *uncertainty in every case of appendicitis*, and it is well to be guarded in every acute case when expressing an opinion; for an apparently simple case may suddenly show dangerous or even fatal symptoms. It is a well-known fact that in the simple catarrhal cases there may never be but one attack, perfect recovery resulting.

Many cases of exudation or abscess, especially of the old cases of so-called perityphlitis, have recovered without operation.

Sahli has collected 7213 cases; of these, 473 were operated on, 6740 were not.

Of the latter, 591 (8.8 per cent.) died; 6194 (91.2 per cent.) recovered; recurrences took place in 4593 cases; of these 3653 recovered without a second recurrence.

Nothnagel claims that 80 per cent. of cases of circumscribed appendicitis recover under medical treatment.

The fact that a patient has recovered under medical treatment from one or two attacks during a period of several years is no guarantee that a fatal issue may not ultimately occur. If a simple catarrhal attack occurs, with moderate symptoms and no marked changes in the blood, the prognosis for immediate recovery is certainly favorable. If no attacks occur during several years, the chances of subsequent attacks are lessened. In all statistics of a second attack and subsequent apparent cure, the history should be investigated for some years. In many cases, when recurrence has taken place, we find chronic appendicitis and practically invalidism as a result.

If in acute cases there are marked blood changes with high polynuclear count, the danger is imminent.

Treatment.—The medical treatment can be summed up very briefly. Absolute rest in bed in the dorsal position. The bowel and urine evacuation should occur with the patient confined to bed.

The physician, having made his diagnosis of acute appendicitis, to the exclusion, of course, of general peritonitis, should pursue the following course: All food and even water at first should be prohibited by mouth. No *cathartic should* at this time be given. Thirst can be relieved by rinsing the mouth with cold water, by small rectal injections of ℥ij to iij (60.0–90.0) of hot normal salt solution at 105° to 108° F., or by proctoclysis. If there is much distention, or if nausea or vomiting, lavage should be carefully performed. In these methods I agree with Ochsner. In the following I differ. If the lavage *does not markedly relieve* the distention (if such be present), I would then advise the physician to personally gently wash the bowel with tube and funnel, after the method of lavage. Only about a quart of normal saline in all at 110° F., need be employed, allowing a few ounces to flow in and then siphoning it out. This practically mechanically carries off the gas and has little influence in producing active peristaltic action. A similar technic is employed in acute distention of typhoid fever with active hemorrhage. He should carefully note the patient's temperature, the rapidity and character of the pulse, and the ratio of pulse to temperature. When feasible, I advocate blood examination in every case, though, of course, it is in some cases impossible. The physician should return to his case in two to three hours for the purpose of further examination. The ice-bag should be applied to the painful area at the first visit.

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[1] American Journal of the Medical Sciences, Nov., 1908.

The pain in almost every case can be controlled by the use of an ice-bag of light weight and a small amount of ice therein, so arranged, supported by a circle of gauze, that only the sensitive area is touched by it. A thin layer or two of gauze can be placed between the bag and the skin, so no damage can be done by the cold, which should be continuously applied. The bag can also be suspended from a barrel hoop to take off the weight, but the former method is the simplest.

In emergency I have used bits of ice tied up in dress shields or in pieces of rubber tissue as a substitute for the bag.

On the following visit or the one thereafter (all of which visits should be made within a total period of nine to ten hours) some definite determination as to the proper course to follow at that specific time can be made. Lavage may again be indicated to relieve distention, or even the special intestinal washing as previously described. Frequently, however, under abstention from food and drink and the application of ice, the tympanites will be markedly relieved and physical examination be comparatively easy. If *there is a history of long constipation*, and on *examination a large fecal accumulation can be determined* in the cecum, I resort to radical treatment.

Fecal accumulation with gaseous distention is an actual danger, from causing pressure on the inflamed appendix. I always resort in such cases to enema or, preferably, gentle intestinal irrigation once or twice a day every day with normal saline solution, with two tubes or a recurrent tube, using 2 to 3 or 4 quarts at each lavage, only ½ pint at a time, at 110° to 115° F., emptying the large intestine mechanically. I have frequently seen the acute symptoms rapidly subside with the above method. These cases are not so very rare.

At the end of forty-eight hours, with symptoms defervescing, a dose of calomel or a saline is then indicated. If there is vomiting, oxalate of cerium or bismuth, and heat to epigastrium; for excessive vomiting, lavage.

The type with fecal impaction is the only class of cases in which such radical methods are resorted to. If in other acute cases in the course of the first ten hours the temperature does not rise markedly, and especially if the pulse does not increase in frequency, but *rather diminishes*, and its character improves, with improvement in the other symptoms, delay, with careful watching of the patient, is allowable. The attack then may gradually entirely subside with no subsequent recurrence or a fresh attack take place after an interval. In the case of a first attack comparatively mild, I would not advise operation subsequent to the attack unless the patient contemplated a journey beyond the reach of a surgeon. Interval operation is always preferable. The indications for surgical intervention are given on page 619.

Diet.—As to diet, I am not quite as radical as Ochsner in all cases. For two days in the acute attack, no matter what the type, I allow nothing by mouth, neither food nor water.[1] The mouth is rinsed and hot salines given by enema or by proctoclysis. After two days I allow small quantities of hot water by mouth, and if the temperature is 100° F. or over, no food by mouth, but nutritive enemata. As soon as the temperature falls to 100° F. or below, food is given by mouth, in small quantities at first. The general diet should then be fluid for some days; no gaseous fluid should be given. Milk well diluted with lime-water or equal parts with barley-water, oatmeal-water, rice-water, gruels, etc., are excellent. Personally, I believe the latter methods preferable to milk, as causing less tympanites; as the symptoms subside, eggs beaten up with milk, bouillon, chicken broth, and later soft-boiled eggs and milk-toast are added.

I agree with Ochsner that lavage is valuable for the distention or for vomiting. If the tympanites, however, is not relieved by the lavage, I believe the funnel method of gentle irrigation of the bowel to be perfectly safe. If fecal accumulation occur later during the attack, a soapsuds enema is indicated. A cathartic should not be given by mouth during the early acute stage until the temperature falls to below 100° F. or until the local symptoms defervesce. Fecal impaction is the exception. Catarrhal colitis should receive treatment as soon as the acute stage of appendicitis has subsided.

Opium has long been a much-vaunted remedy, on the theory of quieting peristalsis and allowing adhesions to form; also for relieving pain; and given preferably as laudanum, or by suppository. I am absolutely opposed to its use. The character of the pulse and respiration are changed thereby; muscular rigidity will relax; and I have seen the symptoms of perforation, both of the appendix and gall-bladder, entirely masked by its employment. Tympanites, distention, and intestinal paresis are more apt to occur as a result of its use. The application of heat I am opposed to.

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[1] This checks peristalsis, as Ochsner claims, and furthermore places the patient in the best condition for operation should such suddenly become necessary.

The ice-bag continuously applied possesses all advantages and no disadvantages; and I only advise the use of a single small dose of morphin by hypodermic, and find it but seldom necessary, if the ice does not control the pain.

Frequent examinations of the blood as regards differential leukocytosis and hyperinosis should be made in every case when possible, at first at least twice daily, and thereafter once a day.

If the differential count is not marked and does not increase, but rather diminishes, and the symptoms gradually defervesce, do not operate during the acute attack.

The indications for operation are as follows: 1. If the patient shows the symptoms of acute peritonitis when first seen or suddenly develops them—general muscular rigidity, tender abdomen, tympanites, etc.—operate immediately.

2. If there be found on examination an area of resistance in the right iliac fossa, and this increases with more marked symptoms after six to twelve hours' observation, whether chills be present or not, operation is indicated.

An aspirating needle should never be employed for purposes of diagnosis.

The blood-count is of value as an aid to prognosis and diagnosis, if the physician has the technical skill or can have it done.

3. In a large abscess, in complicated cases, or when temperature is steadily rising, operate.

4. If the course of the disease is protracted and the symptoms point to abscess or an active and progressive process, operate.

5. In acute fulminating cases. In this type, with apparently mild local symptoms, but especially a gradual increase of pulse and a moderate increase of temperature, there should be immediate operation. The blood examination is important, if possible.

6. A frequent pulse, increasing in rapidity, not corresponding to the more gradual rise of temperature, indicates operation. In all cases when the differential blood-count is marked and increasing, operate.

7. If the patient have a mild attack and subsequently develop a second attack (of less severe type than the first), delay may occur; but if a third attack occur, then an interval operation. If the second attack be more severe than the first, then interval operation.

8. In chronic appendicitis, with symptoms persistent and invalidism, or if recurrent acute exacerbations, operate.

9. In harmful involution of the appendix, operate.

In effect, the best judgment is, operate if possible during interval; and do not operate if one or two mild catarrhal attacks.

The method of operation depends on the location and type of appendicitis.

CHAPTER XXX

DIVERTICULITIS—PERIDIVERTICULITIS

(*Synonyms.*—Sigmoiditis; Perisigmoiditis.)

History.—During the past fifty years specimens of false diverticula of the descending colon and sigmoid, both with and without concretions, have been reported by pathologists, who have demonstrated their relationship to general or local peritonitis. Only recently has attention been especially focused on inflammation in the left iliac fossa, and the terms "sigmoiditis" and "perisigmoiditis" been employed.

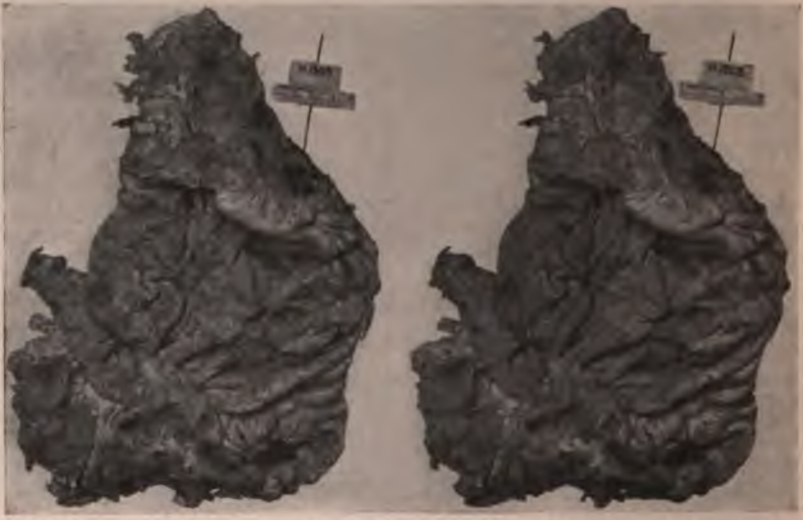

Fig. 223.—Diverticulitis. Sigmoid laid open longitudinally. A diverticulum containing a sloughing ulcer is seen at the lower right hand; another is sectioned near the label needle (W. J. Mayo).

There is confusion as to the definition of sigmoiditis, many using it in the sense of a catarrh of the sigmoid; while others define it as an inflammatory condition, involving to a greater or less degree this entire portion of the gut (the *musculature included*). In other words, it is not a catarrh of the mucous membrane. It was so defined as a diffuse inflammation by Patel[1] and will be so employed in this volume. Eisendrath[2] has recently contributed to this subject.

Diverticulitis[3] and peridiverticulitis, as when productive of disturbance they occur chiefly in the sigmoid, have been used interchangeably with sigmoiditis; and perisigmoiditis bears the same relationship as does perityphlitis to typhlitis.

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[1] Revue de Chirurgie, Oct. and Dec., 1907; Lyon Med., Oct. 2, 1905.

[2] Specific inflammations, such as dysentery, tuberculosis, and syphilis, are excluded (author).

[3] Sigmoid Diverticulitis, Archives of Diagnosis, Oct., 1909.

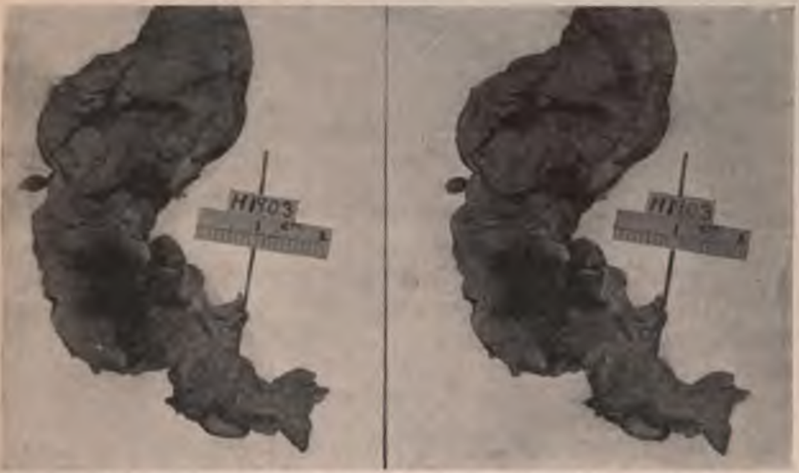

Fig. 224.—Diverticulitis. Section through ulcerated diverticulum shown in Fig. 223 (W. J. Mayo).

Mayo first employed the term sigmoiditis, but the condition was first described by Joseph M. Mathews. J. P. Tuttle[1] has recently contributed an excellent monograph. Though inflamed diverticula of the sigmoid undoubtedly cause the maximum of all cases of sig-

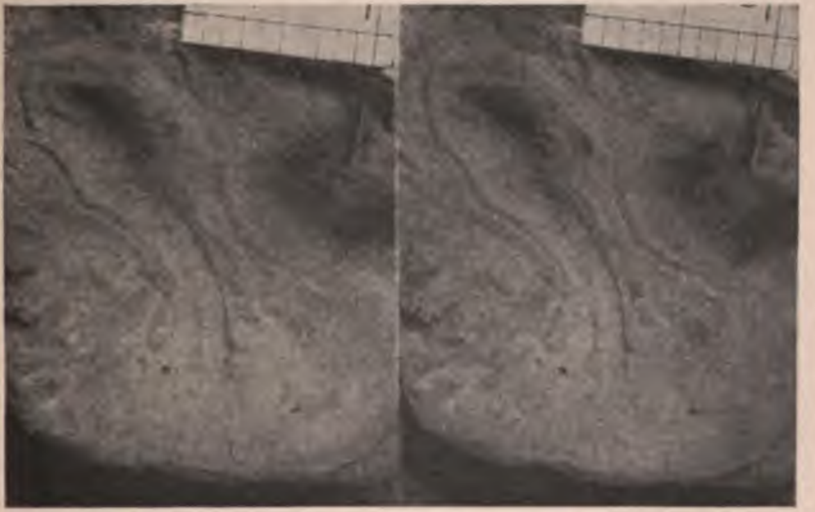

Fig. 225.—Diverticulitis. Enlarged view of sectioned diverticulum shown in Fig. 223. Note muscularis in wall, occluded lumen, and inflamed submucosa (W. J. Mayo).

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[1] American Journal of Surgery, April, 1909.

moiditis and perisigmoiditis, having about the same relation to left iliac abdominal suppuration as the appendix has to similar conditions in the right iliac fossa, yet other causes of perisigmoiditis are given, such as ulcerations extending through the wall of the gut; traumatism or puncture by foreign bodies; diverticula, in association with which Byron Robinson and Tuttle[1] believe that *traumatism*

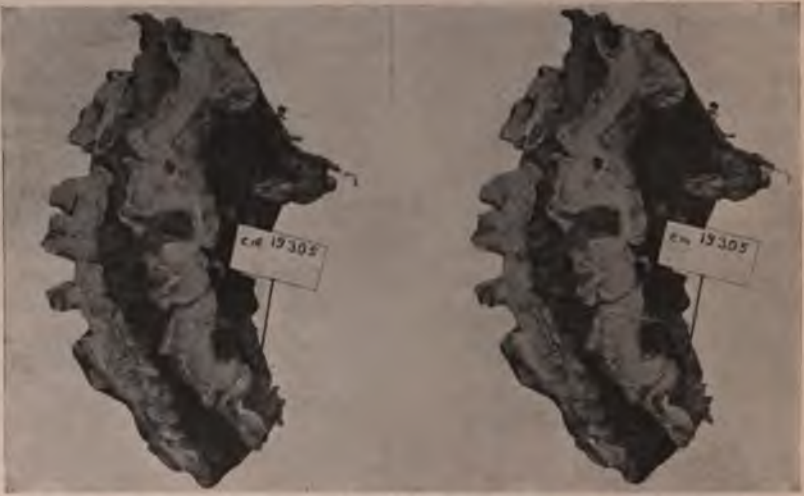

Fig. 226.—Peridiverticulitis. Sigmoid divided longitudinally. Note defective musculature and the diverticula. Inflammatory mass dissected away near label needle (W. J. Mayo).

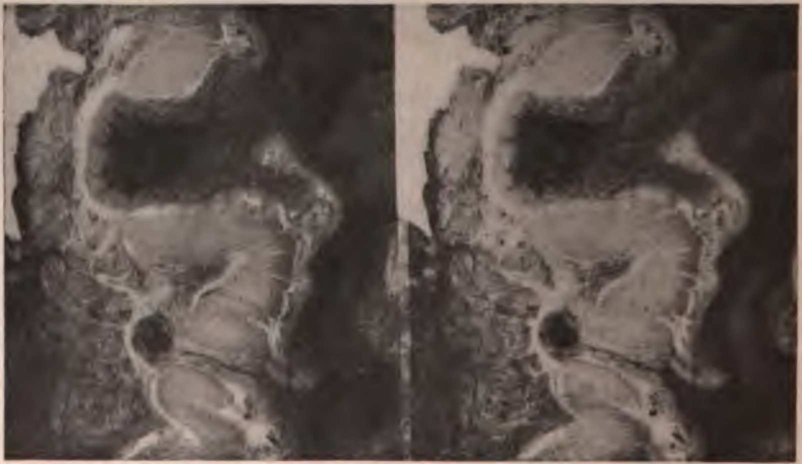

Fig. 227.—Peridiverticulitis. Enlarged view of two diverticula and one point of defective musculature seen in Fig. 226 (W. J. Mayo).

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[1] American Journal of Surgery, April, 1909 (J. P. Tuttle).

from the iliac and psoas muscles play a part; lumbricoid worms and wisps of hay entering the appendices epiploicæ. Secondary perisigmoiditis is also believed to occur from inflammation by extension from other abdominal or pelvic organs.

Wm. J. Mayo[1] draws a shårp distinction between diverticulitis and peridiverticulitis (Figs. 223–228).

With diverticulitis there is a primary lesion in the mucosa, often of ulcerative type, and a tendency to perforation into the peritoneal cavity, with resulting acute peritonitis. It has no tendency to produce reduction of the lumen of the bowel.

With peridiverticulitis there is a leakage of toxins and bacteria into the subserosa and tissues surrounding the diverticulum, with resulting inflammation and thickening of the gut, so that its lumen may be markedly reduced and symptoms of obstruction occur. Perforative peritonitis rarely occurs in this type, as so much reparative inflammation goes on. Local intraperitoneal abscess or acute or chronic obstruction may result.

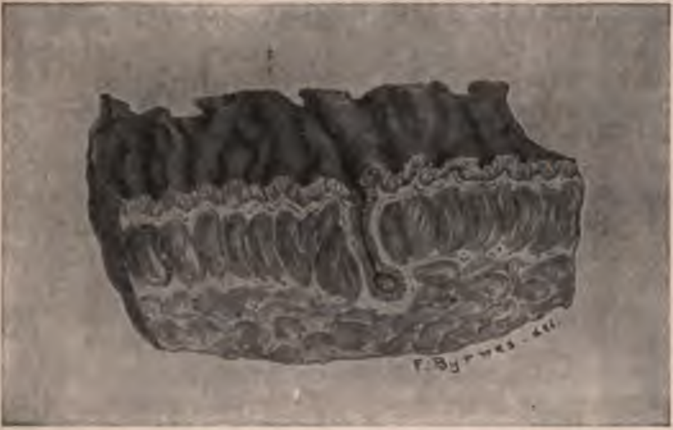

Fig. 228.—Peridiverticulitis. Sketch of diverticulum with inflammatory deposit in subserosa; 11 diam. (W. J. Mayo, Wilson, and Giffin).

Lejars,[2] Bittorf,[3] and Rosenheim[4] classify sigmoiditis and perisigmoiditis clinically. Gordinier and Sampson[5] hold that these conditions are more frequent than we suppose.

Diverticula of the Intestines.—Diverticula are formed by a bulging or protrusion of the intestinal wall, and are divided into the congenital and the acquired.

In the congenital forms the wall of the diverticulum is formed by the whole intestinal wall; and these have been called "true" diverticula. It was formerly thought that all acquired diverticula were of the "false" type, consisting of protrusions of the mucosa through spaces in the muscular coat, so that their wall comprised mucosa and serosa. It has been demonstrated that acquired diverticula may be of the "true" type, and are caused most frequently by traction from tumors or adherent organs. False diverticula are the result of excessive pressure within the intestines combined with a congenital weakness of the bowels. The chief congenital diverticulum of importance is Meckel's.

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[1] Surgery, Gynecology, and Obstetrics, July, 1907.
[2] Semaine Medicale, June 27, 1904, p. 26.
[3] Münchener med. Wochenschr., 1904, p. 147.
[4] Zeitschr. für Klin. Med., 1904, Band. liv, p. 475.
[5] Jour. Am. Med. Assoc., 1906, vol. i, p. 1686.

Meckel's diverticulum, due to the persistence or incomplete obliteration of the omphalomesenteric duct, usually rises from the ileum ½ to 1 meter above the ileocecal valve, from the convex margin of the intestines opposite the mesenteric attachment, and varies in length from 3 to 10 cm., though rarely longer.

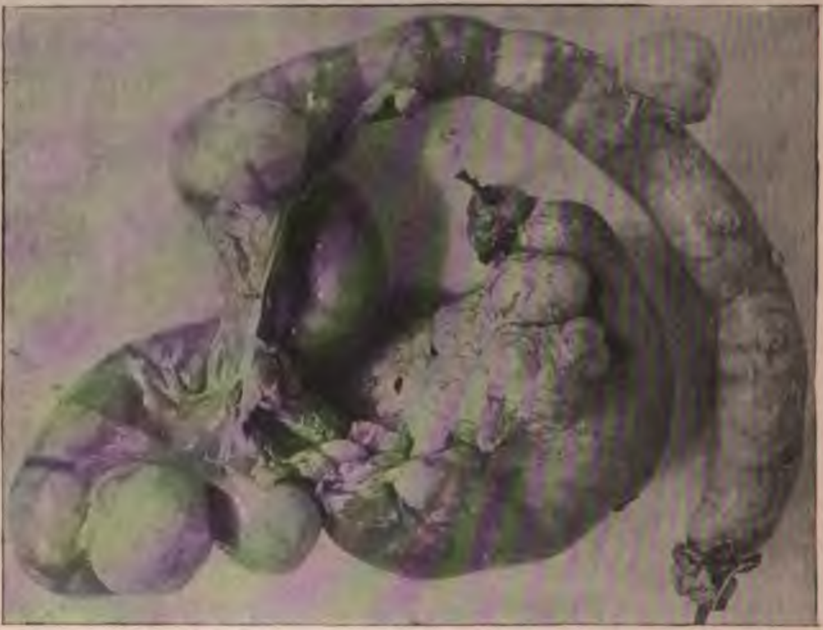

Fig. 229.—Hodenpyl's specimen of multiple acquired diverticula of the colon (sigmoid flexure) (Brewer).

Congenital diverticula have been found in the small and large intestines.

Acquired diverticula occur both in the small and large intestines, and have even been recorded in the appendix by Edel[1] and Mertius.[2]

They are generally more frequent in the large intestine, especially in the lower part of the descending colon and sigmoid flexure, the latter providing the most examples, as in Fig. 228, reported by G. Brewer.[1] Here they are usually multiple.

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[1] Virchow's Archiv., Bd. cxxxviii.

[2] Mitheilung ans der Grenzgebiet für Med. und Chur., Bd. ix.

Autopsies in death from other causes have been reported where diverticula were found, but no symptoms had ever occurred. As many as 400 diverticula in one case have been noted by Hausemann.

One case of diverticulitis of the small intestine which gave symptoms has been recorded by Gordinier and Sampson.[2]

With diverticula of the appendix, appendicitis has been simulated, but it is clinically unimportant to separate the conditions, as operation is indicated in any event. Diverticula are most frequent in the sigmoid flexure. Quite a number give important symptoms. E. Beer[3] has described the clinical symptoms of diverticulitis and Telling[4] has collected 105 cases, giving a thorough exposition of the subject.

Sudsuki[5] found diverticula present 15 times in 40 autopsies, so that the condition is probably more frequent than has been supposed, and has often been overlooked, even in post mortem.

Brewer[6] has reported a case of acute diverticulitis of the sigmoid, with operation before rupture.

Occurrence.—Acquired diverticula are much more common in the large intestine, more so in the descending colon, and especially so in the sigmoid flexure. They are usually multiple and may arise from any part of the surface. They are frequently seen in two rows at the sides of the gut, or *close to the mesenteric attachment,* more rarely on the convexity.

Chlumsky claims that he finds by experiment that rupture in the living bowel upon distention occurs more frequently opposite the mesentery. The general opinion is that the mesenteric side is less resistant. Probably the most common occurrence of these diverticula is in the appendices epiploicæ. In many cases they are confined to them, and present a double row of symmetrically placed hollowed-out pockets. In other cases none are so situated, and frequently some enter the appendices and some lie outside of them. The presence of the diverticula in these appendices may account for some of the tenderness found in many portions of the gut. The special favoring of the epiploicæ is accounted for by the fact that *the point of their attachment to the gut is a place of least resistance.*

In a majority of cases the affected appendages have been or are filled with a large amount of fat.

Bland Sutton[7] has stated and illustrated by diagrams that this fat is directly continuous with the subserous fat. If there is the slightest tendency to the formation of diverticula, it will readily be seen that the soft fatty tissue of the appendices epiploicæ form a point of lowered resistance.

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[1] Am. Jour. of Med. Sci., Oct., 1907.
[2] Jour. Am. Med. Assoc., 1906, vol. i, p. 1684.
[3] Am. Jour. of Med. Sci., July, 1904.
[4] Lancet, March 21 and 28, 1908.
[5] Langenbeck's Archiv., Bd. lxi, p. 708.
[6] Jour. Am. Med. Assoc., Aug. 15, 1908.
[7] Lancet, Oct. 24, 1903, p. 1148.

Size.—The diverticula vary in size from a millet-seed to a hazelnut. Large size is seldom attained, as secondary changes occur leading to detachment, ulceration, abscess, or peritonitis.

When small, they are semiglobular; as they increase in size, more oval or flask shaped; the aperture on the gut wall usually being smaller than the maximum diameter of the diverticulum, and almost constantly so when they enter the appendices epiploicæ.

This is an anatomic point of great importance in regard to the causation of inflammation. In those which do not enter the appendices the aperture may be relatively large. They are then generally true diverticula and are formed from normal haustra (Sudsuki),[1] usually not extending much above the middle of the descending colon. They increase in number and size from above downward, and may be quite crowded together in the sigmoid flexure. At the commencement of the rectum they generally stop abruptly. This is possibly due to the absence of appendices epiploicæ in this situation; but the fact that the feces are not retained so long in this part of the bowel is also partly responsible. Schreiber[2] thinks that the stronger musculature of the rectum plays some part in preventing their formation.

They are almost invariably filled with fecal material, generally concretions of some degree of firmness. This fact probably determines their subsequent clinical importance, and sharply distinguishes them from *acquired diverticula in the small intestine, which rarely contain fecal material.* To this we can ascribe the immunity of the latter from secondary pathologic processes and symptoms.

Usually in the early stages all the coats of the bowel are represented, but in some the muscularis is absent. There has been controversy as to the presence or absence of muscular fibers in the diverticulum wall. Cruveilhier and Rokitansky originally regarded the diverticula as hernial protrusions of the mucous and serous coats, with absence of muscular tissue. The presence or absence of muscle was for many years the criterion for distinction between congenital and acquired diverticula.

The microscopic findings in numerous cases show that acquired diverticula often *have all the coats of the bowels represented.* In the early stages of the formation it is almost the rule, although the muscular tissue undergoes atrophy as the sac enlarges.

Etiology.—One[3] case has been so far recorded in a child, while the great majority are in old or even aged subjects. They seem to be generally acquired.

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[1] Langenbeck's Archiv., Band. lxi, p. 708.

[2] Deutsche Archiv. für Klin. Medicin, 1902, p. 122.

[3] In a child seven years of age an acute catarrhal inflammation of diverticula in the sigmoid occurred (Annals of Surgery, vol. xlvii, by A. P. C. Ashhurst).

1. Generally, advanced age of the patient. In 80 cases, average age sixty years, but of those in whom diverticula caused symptoms in 47 cases, the age was fifty-five years. In 33 cases no symptoms were referred to their presence, but they were accidentally discovered, the age was sixty-seven. Fiedler[1] records the youngest case at twenty-two years. William J. Mayo places the majority at over fifty years.

2. *Sex.*—In 81 cases—53 males, 28 females.

3. *Obesity.*—Klebs, Mayo, and others have laid stress on this. Many have been fat; the presence of much fatty tissue in the gut walls and appendices epiploicæ have been shown by Bland Sutton to predispose to mucosal out-pushings. In 22 cases, 17 were stated to be more or less obese and to have much fatty tissue in the gut wall.

4. *Cachexia and Absence of Fat.*—On the other hand, many subjects are noted as having been thin (Hausemann)[2]; 5 of the 22 cases were stated to be of this type. Undoubtedly some had previously been obese, and from age or illness had lost much of their fatty tissue. This has not been definitely referred to. It will be readily understood that the fatty deposit would first weaken the gut wall; the subsequent loss of fat with possible concomitant weakening and wasting of the muscle fibers would probably increase this tendency.

5. *The Normal Structure of the Large Intestine.*—This readily lends itself to local yielding of its walls, as is suggested by the *normal sacculi, which are often exaggerated in cases of constipation,* sometimes in association with diverticula.

6. *Physiologic Rôle of the Sigmoid Flexure.*—In this portion of the bowel is *the longest retention of fecal material,* and here consequently *the pressure from within will be liable to be the greatest.*

7. *Pressure from Within the Bowel.*—This may be due to accumulation of feces or gas, or both.

(*a*) *Presence of Constipation.*—This stands in *most frequent and important causal relationship to this condition.* In 22 cases in which the point is definitely mentioned, constipation, often severe and generally for a considerable period, was present in 17. In the remaining 5 its absence was particularly noted. Constipation in old people, whether obese or emaciated, *is very common,* while diverticula are comparatively rare; hence, other factors must be present.

(*b*) *Flatulence.*—This is usually associated with constipation, and acts in the same way by *increasing internal pressure;* so much stress has been laid on the factor of internal pressure that the term "*pulsion diverticula*" has been applied to them.

8. *The Relation of the Diverticula to the Points of Entry of the Vessels through the Gut Walls.*—This fact was first pointed out by Klebs[3] in the case of acquired diverticula in the small intestine, and

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[1] Denkschrift der Gesellschaft für Natur, v. Heilknade, Dresden, 1868.

[2] Virchow's Archiv., Band, cxliv.

[3] Pathologic Anatomy, 1869, p. 271.

has been confirmed by others (Hausemann and Fisher) with regard to the large intestine.

Microscopically it is evident that *the spots in the gut wall* where it is pierced by the *vessels are areas of weakened resistance to internal pressure*, because the vessels are accompanied by a certain amount of lax connective tissue, through which an out-pushing of the mucous membrane can more easily take place.

9. *Variations in the Size of the Vessels.*—Graser[1] was the first to investigate this point. In his case, the patient suffered from chronic heart disease, with venous back-pressure, leading to distention of the veins in the gut wall. This dilatation he regarded as further weakening the vessel spaces by pushing aside the muscular fibers. He examined microscopically the sigmoids of 28 patients who had suffered prior to death from mesenteric venous stagnation. In 10 of them he found definite evidence (mostly microscopic) *of commencing out-pushings of the gut wall;* and in every case they occurred through these "vessel holes."

The diverticula were most numerous in the sigmoid and practically ceased at the middle of the ascending colon.

Mesenteric venous congestion may be due to chronic heart or lung disease, portal back-pressure, or intra-abdominal tumor, etc.

"He is inclined to ascribe a special importance to a distention which is not constant, but of frequent repetition, as recurs in the repeated failures of cardiac compensation and in frequent recoveries therefrom in patients with chronic heart disease."

When the vessels are engorged the vessel holes are enlarged; when they are smaller these areas are thereby weakened, and there is a *greater liability to hernia of the mucosa.*

While in the small intestine the diverticula are practically always on the mesenteric side of the bowel where the vessels pierce the walls, *in the larger bowel* many of the diverticula occur on *the side remote from the mesentery*. Another explanation must be sought for.

Therefore, Schreiber believed the congestion of the vessels was secondary to the presence of feces in the diverticulum, rather than to the original cause of the formation of the latter.

10. *The Connective Tissue Around the Vessels.*—Sudsuki[2] found in 40 cases not suffering from mesenteric venous stagnation which he examined microscopically, diverticula present in 15 bodies; in 6 cases, true diverticula, that is, all the coats were therein. In 20 cases, where mesenteric congestion was present, he found diverticula in only 6; in 12 cases free from congestion, diverticula were found in 9.

The subjects were all adults and nearly all men, middle aged or old. He suggests there is *a congenital predisposition with regard to the amount and laxity of connective tissue surrounding the vessels at these spots;* if there is much fatty deposit, this will act in the same way;

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[1] Centralblatt für Chirurg., 1898, etc.

[2] Langenbeck's Archiv., Band. lxi, p. 708.

and if there be subsequent wasting of such fatty tissues, *further weakening takes place.*

Beer states that this theory fails to explain the non-mesenteric diverticula and those which have muscular layers in their walls. These vessel spaces have some influence, but they have some additional cause, and Beer finds this in the following:

11. *Muscular Deficiency of the Gut Wall.*—Since diverticula occur in old people, in whom the muscular power of their intestines has been more or less exhausted (as evidenced by constipation), and are in association with obesity (or obesity followed by cachexia), these facts all point to a muscular deficiency.

In this muscular weakness Beer thinks the cause of the false diverticula must be sought.

In Mayo's cases areas of muscular deficiency were noted opposite early diverticula, or even in areas yet free from out-pushing. Probably no one factor is sufficient.

Out of 105 cases reported, 60 per cent. were attended with symptoms (Telling).

Secondary Pathologic Processes in the Diverticula.—The diverticula of themselves occasion no symptoms, but, as one would expect, readily undergo inflammatory changes therein. They tend in most cases to form fusiform pouches connecting with the lumen of the *gut by a constricting neck*, and these pouches are situated for the most part on that portion of the bowel—the sigmoid flexure—of which the normal anatomy and physiology favor most the retention of feces and the accumulation of gas. If a condition of constipation exists and the "force from within" is increased, it is almost inevitable that they will from the first have fecal contents.

On account of the narrow neck and deficient muscular fibers in the sac wall the contents rarely are expelled and concretions form. These probably cause the trouble. The first result would be a tendency to enlargement of the sac. Then the muscular layers atrophy, as do the glands; the muscle may be replaced by fibrous tissue. The increase in the size of the diverticulum, with atrophic changes in its walls, produces necessarily a dangerous thinning of the sac. In some cases there is little more than a peritoneal covering, with *the contained feces visible through it.* The irritation of the retained and hardened feces then leads to inflammatory changes. These may be slight and only microscopic in the mucosa and submucosa, or may produce *more serious acute or chronic lesions.* The fecal matter is a nidus for bacteria; their products undoubtedly determine the nature of the inflammatory reaction which occurs. An ulceration may result from bacterial infection. Moreover, the concretion will tend to be forced through an inflamed or ulcerated area. These features explain the occurrence of local abscess or general peritonitis. The latter may be also the result of sudden trauma or strain, which may cause an increased pressure within the bowel.

Definite types are produced by the following causes (Telling):

1. Thinning of the diverticulum wall.
2. Perforating action of the retained concretion.
3. The presence of micro-organisms and their toxins.
4. Inflammatory reaction of varying types and degree.

With these data[1] one can forecast the various cases which one might expect clinically; viz.:

1. Infection of the general peritoneal cavity from thinning of the sac walls without perforation.

2. Acute or gangrenous inflammation—"diverticulitis."

3. Chronic proliferative inflammation, with thickening of the gut wall and stenosis of the bowel.

4. Formation of adhesions, especially to the (*a*) small intestine, (*b*) bladder.

5. Perforation of diverticula, giving rise to (*a*) general peritonitis, (*b*) local abscess, (*c*) submucous fistulæ of the gut wall, (*d*) fistulous communication with other viscera, especially the bladder.

6. The lodgment of foreign bodies. 7. Chronic mesenteritis of the sigmoid loop. 8. Local chronic peritonitis. 9. Metastatic suppuration. 10. Development of carcinoma. 11. Perforation into a hernial sac.

The following classification, by Telling, I believe the most scientific:

1. *Infection of the general peritoneum* as a result of thinning of sac walls. Organisms make their way through the wall and cause peritonitis *without perforation*. Loomis[2] records 1 case.

2. *Acute Gangrenous Inflammation of the Diverticulum.—Symptoms.*—Pain, tenderness, and swelling in the left inguinal region are present. Local abscess or general peritonitis may result. Rigidity of the left rectus; fever; hyperinosis (increased fibrin in the blood); leukocytosis; increase in polynuclears are present. If general peritonitis, we have the additional symptoms.

3. *Chronic Inflammation.*—A chronic proliferative inflammation of the submucous and serous coats may occur. Thickening may be considerable. It may lead to (*a*) tumor formation, (*b*) *stenosis with obstruction*, (*c*) *mimicry of carcinoma*.

This type of inflammation is most important, most frequent, and generally overlooked. Grasser[3] in 1898 first described a case. Moynihan recorded one. The mimicry of carcinoma is so perfect that not only is the diagnosis made during life but also at the operation, and again confirmed erroneously at post mortem.

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[1] Eisendrath gives a somewhat simpler clinical classification:

1. Acute: (*a*) Acute catarrhal; (*b*) acute gangrenous, with or without local abscess; (*c*) acute perforative, with general peritonitis.

2. Chronic: (*a*) Chronic hyperplastic or stenosing (peridiverticulitis); (*b*) enterovesical fistulous form; (*c*) chronic adhesive form, causing acute or chronic obstruction.

[2] New York Medical Record, 1870, vol. iv.

[3] Centralblatt für Chirurg., 1898.

Differential diagnosis between carcinomatous stenosis and diverticular stenosis is as follows:

With *carcinoma* there are nearly invariably an involvement and ulceration of the mucous membrane, with fungation of growth into the lumen of bowel. With *diverticulitis* it is the rule for the mucous membrane to be free from ulceration (unless a fistulous tract or abscess cavity open into it from without inward); also the folds of the mucosa are strongly marked and crowded together, giving an unduly rugose appearance. The orifices of the pouches may be visible, but are generally small and often concealed by these folds. One should examine the folds with a fine probe. There is usually *absence of blood and pus* in the stool in *diverticulitis*, but *present* in *carcinoma*.

Age of patients the same.

Stenosis may cause acute or chronic obstruction (Mayo).

4. *Formation of Adhesions.*—(*a*) Adhesions to small intestine may produce acute or chronic obstruction. (*b*) Adhesions to bladder are also noted.

5. *Perforation of Diverticula.*—Results differ, according to (*a*) acuteness of ulcerative process, (*b*) amount of chronic inflammatory thickening present, (*c*) presence of adhesions.

These factors determine whether perforation leads to (*a*) general peritonitis, (*b*) local abscess formation, (*c*) fistulous communication with other viscera, notably the bladder.

(*a*) *General perforative peritonitis* has occurred in 14 cases. C. A. McWilliams reported a case at the Surgical Society, October 30, 1907, which he operated upon at the Presbyterian Hospital for general peritonitis; the history of constipation of only a week's duration. Male, age forty-seven. The case was believed to be perforative appendicitis, not having been seen *until general peritonitis was in evidence.*

Milky fluid under considerable tension was evacuated at operation. There was also fluid between the liver and diaphragm. Appendix, gall-bladder, stomach, and pancreas were examined, but no perforation was found.

The patient's condition became such that further search was deemed inadvisable. Post mortem showed diverticula of large size commencing in the ascending colon. In the descending colon, 10 cm. below the splenic flexure, a perforation of a diverticulum was discovered. Culture from the peritoneum, etc., showed Bacillus coli.

(*b*) *Local Abscess Formation in 24 Cases.*—There may be several small abscesses shut off by thick adhesions or a single large abscess. The abscess may be intra- or extraperitoneal, and may lead in turn to communication with the external surface, with the bladder or bowel.

(*c*) *Submucous Fistulæ.*—In some cases with much inflammatory thickening the ulceration of the inside of the sac leads to a small abscess. This happens in several foci in the thickened area. These abscesses tend to burrow through the thickened tissues and form *submucous fistulous* communications with each other. There may be a labyrinth of such tracts. They may re-enter the lumen of the sigmoid or communicate with a peritoneal abscess-cavity by one or several openings. This suppurating process is often excessively chronic and gives rise to *great thickening*, adhesions, and tumor formation, with sequelæ in the shape of intestinal obstruction or adhesions to and subsequent perforation of the bladder.

6. *Lodgment of Foreign Bodies.*—Diverticula are liable to harbor foreign bodies, which may give rise to perforation, diverticulitis, or local abscess. Bland Sutton reports several cases.

7. *Chronic Mesenteritis.*—In some there is inflammatory thickening of or abscess formation in the sigmoid mesentery. It is believed by Ries[1] that retraction of the sigmoid loop is produced thereby and also adhesions, and hence *twists*, *kinks*, or *volvulus*.

8. *Local Chronic Peritonitis.*—This is often found in the neighborhood of the sigmoid, causing thickening, opacity, or adhesion of the peritoneum, probably in some cases due to leakage of toxins through the thin wall of an overlooked diverticulum, though at times due to the pelvic organs.

9. *Metastatic Suppuration.*—One case recorded, with abscesses of the liver from diverticular abscess (Whyte).[2]

10. *Development of Carcinoma.*—Secondary to diverticulitis.

Carcinoma may result from chronic irritation and ulceration of diverticula due to retained feces (Hochenegg[3]).

11. *Perforation of a Hernial Sac.*—One case recorded (Stierlin[4]).

Clinical Aspects.—Patel,[5] in discussing inflammation of the sigmoid, classifies these conditions as acute non-suppurative sigmoiditis, suppurative perisigmoiditis, chronic perisigmoiditis, sigmoiditis, and sigmoiditis with general peritonitis.

Inflammatory trouble, more or less acute in the left lower abdomen, has been described as left-sided appendicitis, sigmoiditis, perisigmoiditis, epiploicæ appendicitis, diverticulitis, or peridiverticulitis.

Clinically, the acute cases resemble appendicitis, except for the fact that the inflammation is on the left side. The symptoms are as follows:

Pain; constipation; tenderness on pressure; muscular rigidity, especially of the left rectus, and later tumor; local tympanites is present. Tumor is not always present.

Left-sided Tumor and Abscess Formation.—In 63 cases tumor was felt in 20, and abscess formation occurred in 23. In some cases with considerable pus formation there may be but little pain and no pyrexia (Georgi).

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[1] Annals of Surgery, vol. xi, p. 523.

[2] Scottish Medical and Surgical Journal, 1906.

[3] Verhandlungen der Deutsche Gesellschaft für Chirurg., Thirty-first Congress, 1902, p. 402.

[4] Correspondenzblatt für Schweitzer Aertze, 1902, vol. xxxii, p. 749.

[5] Revue de Chirurgie, Oct. 10, 1907, and Dec. 10, 1907.

Tumor is *elongated, sausage shaped, tender, and often ill defined.* It may be movable or fixed, lying a little above Poupart's ligament (frequently parallel with it).

Shortly after the occurrence of symptoms, this swelling may be made out; it may disappear rapidly or gradually, or may increase with signs of pus formation. Patel has shown that the tumor may be higher, lower, or posterior. The position depends on the location of the inflamed diverticulum. In a patient of middle age or older, diagnosis of diverticulitis would be probable.

Differential diagnosis must be made from appendicitis, with left-sided symptoms,[1] pelvic inflammation, ovarian cyst, with inflammation or strangulation, actinomycosis of the sigmoid, syphilitic or tuberculous pericolitis, sigmoid catarrh, and dysentery. Tuberculosis is too often accepted.

Intestinal Obstruction.—When acute, this is probably due to secondary involvement of a coil of small intestine and is not to be *diagnosed except at operation.*

The same is true of chronic obstruction of the small intestine. Obstruction, which is chronic, recurrent, or acute engrafted on chronic, when localized in the sigmoid region is nearly always diagnosed as the result of carcinoma, when it occurs in an elderly person with a history of constipation.

Advanced age, recent constipation, cachexia, and blood in the stools favor cancer; while long-standing constipation, absence of blood in the stools after repeated examinations, and slight cachexia (or rather loss of weight), together with an evidence of pus formation, would favor peridiverticulitis.

Entire absence of constipation may occur with either, and blood has been found in one diverticular case. Probably many of the so-called cases of cancer are really this condition.

Perforative Peritonitis.—In these cases a routine examination of the sigmoid should be made.

Vesicocolic Fistula.—Probably many of these cases, supposedly due to cancer of the sigmoid, are the result of diverticulitis with adhesions and perforation of the bladder. Air and feces pass through the urethra. Cripps found 45 cases of vesico-intestinal fistula out of 63 to be inflammatory, only 9 cancerous, and some of these doubtful.

There seems to be a liability of postoperative peritonitis following an operation for diverticulitis, probably from other thin-walled diverticula.

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[1] An appendix passing transversely to the left iliac fossa can sometimes be determined by vaginal or rectal examination. An abnormal position of the cecum occasionally occurs. With tuberculosis, bacilli may be found in the stools, also pus and blood, but not always at first. The tuberculin reaction, ocular or by injection, aids diagnosis. Pus and blood appear in dysentery and syphilis, and the Ameba or Bacillus dysenteriæ in the former and Wassermann's reaction in the latter. Mucus alone is present in catarrh. Actinomyces are found in the stool in actinomycosis. Vaginal examination helps diagnose pelvic conditions.

Diagnosis.—To recapitulate: Diverticulitis occurs usually in persons over thirty-five years of age suffering from constipation and of obese habit. The site of pain about corresponds on the left side to that of appendicitis on the right. Muscular rigidity, especially of the left rectus, is present. There is tenderness on pressure in the left iliac fossa. Tumor may or may not be present.

In suppurative cases there may be chills. Leukocytosis is present. The increase in the polynuclears and hyperinosis are of importance for diagnosis.

The various types have been described; among the most common are acute peritonitis, resulting from perforation; local abscess, which may break into some viscus, as the bladder, or which must be opened; acute or chronic obstruction, resulting from stenosis of the sigmoid from peridiverticulitis; chronic tumors, simulating carcinoma; and milder cases, with local pain, tenderness, constipation; and a local tumor, which symptoms gradually subside under treatment.

Treatment.—The acute cases should receive the same treatment as acute appendicitis. In mild cases the bowel should be emptied by enema; liquid diet, preferably broths and gruels; ice-bag continuously applied; absolute rest in bed. Subsequent *attention, after recovery*, should be directed to the careful regulation of the bowels by diet and medication. Intestinal irrigation and high olive oil injections also are of value. Cases of chronic stenosis may be temporarily treated as such. Indications for operation are the same as in appendicitis.

Acute obstruction, peritonitis, and abscess require immediate operation. Chronic obstruction may require resection. Appropriate operative procedures are necessary for the complications described.

CHAPTER XXXI

INTESTINAL OBSTRUCTION—ACUTE AND CHRONIC

INTESTINAL obstruction occurs in two types—acute and chronic.

Acute obstruction is, in turn, characterized by two anatomic types:

1. Acute intestinal obstruction with sudden complete occlusion of the intestinal lumen.

2. Acute intestinal obstruction engrafted suddenly on a chronic obstruction (chronic stenosis), due to a sudden blocking of the stenosed intestines from various causes, such as by a foreign body or fecal accumulation above the stricture.

ACUTE INTESTINAL OBSTRUCTION

(*Synonyms.*—Ileus; Miserere; Passio Iliaca.)

General Considerations.—Acute intestinal obstruction may be defined as a sudden acute stoppage of the passage of the intestinal contents. This may be caused by mechanical occlusion of the intestinal canal (mechanical ileus), by a sudden loss of motor power in a portion or in all the bowel (dynamic or paralytic ileus), or by a combination of these conditions.

Before discussing the matter further, as a means of assistance to diagnosis, I wish to call a few very important facts to my readers' attention.

As a rule, we may say that acute obstruction of the small intestine gives rise to more severe and violent symptoms than that of the large intestine. The nervous apparatus of the small intestine is in connection with most important plexuses, the solar and superior mesenteric, and the pain is more violent, the vomiting earlier and more marked, and the prostration and shock more rapid.

The early appearance of indicanuria and of anuria are also significant of obstruction in the small intestine.

Moreover, simple occlusion of the intestines does not lead to nearly as acute symptoms as when strangulation is present. In the latter condition interference with the circulation of the intestinal wall and of the mesentery and the irritation of the sensory nerves give rise to acute and violent symptoms, marked pain, vomiting, and shock. In this class of cases we have the acute internal hernias and strangulations, volvulus, and the severe type of acute intussusception. Local meteorism is generally present in the early stages of volvulus, internal herniaform strangulation and kinking of the bowels, and enables us at times to locate the character of the lesion. Often this distention rapidly becomes very extensive, as in volvulus, for example, a general distention ensues, so that determination of the condition is difficult.

In the majority of cases of acute obstruction when we find present marked increased peristaltic movements of the bowel, with stiffening and rigidity of loops of intestines, they occur in acute cases engrafted on cases of chronic stenosis with hypertrophy of the bowel muscle above the stricture. We occasionally see this symptom in primary acute conditions, especially in acute intussusception.

We must remember, moreover, that acute dilatation of the stomach, either alone or associated with intestinal paresis, quite frequently occurs after operation, and presents many of the symptoms of acute dynamic ileus. There is obstruction, in fact, in many cases due to pressure of the organ on the transverse duodenum or, as some believe, by mesenteric traction. The stomach may occupy the left half or even the entire abdominal cavity. I have referred to this condition under Acute Dilatation of the Stomach.

Practically we may say that in every case of acute obstruction intra-abdominal tension is markedly increased, thus interfering with the physical examination. I place the following suggestions at the commencement of this chapter in order to emphasize them the more. They are of value as an aid to diagnosis in every case with acute symptoms:

Immediate and thorough lavage, digital examination of the rectum, and if no evidences therein of obstruction or intussusception, preventing the entrance of an injection, then a careful recurrent rectal irrigation; vaginal examination and inspection of hernial openings should be the preliminaries in the examination of every case. Lavage and irrigation of the bowel immediately promote the comfort of the patient by lessening distention, render the physical examination easier, and the combined methods are an invaluable aid in diagnosis. It may be necessary to substitute a high enema for irrigation.

Etiology of Acute Intestinal Obstruction.—The various causes of acute intestinal obstruction on account of their importance will now be considered separately:

The first classification that I shall describe is the so-called *internal herniaform strangulation of the bowel* (compression of the intestines), due to strangulation by (*a*) bands and adhesions, the result of a former peritonitis; (*b*) Meckel's diverticulum; (*c*) slits and apertures; (*d*) incarceration into herniæ; (*e*) tumor pressure from without.

Frequency.—Fitz, in an analysis of 295 cases of acute obstruction, gives 34 per cent. of the cases as due to this type of strangulation (internal herniaform), excluding volvulus; 35 per cent. out of 1134 cases are reported by Leichtenstern.

Out of 101 cases of strangulation Fitz shows that 63 were due to adhesions and bands, and 21 to vitelline remains.

Strangulation of the intestines by adhesions and bands thus constitutes the largest percentage of this class. In some cases they may be congenital. The band may be a firm fibrous cord, or may be tough and thin as a thread, occasionally it may be ½ inch wide.

Strangulation from bands and adhesions may occur in four ways:

1. There may be a short tense band firmly attached at each end, beneath whose arch a knuckle of the intestines passes, the space may only be of a size to admit two to three fingers.

2. On the other hand, there may be a long lax band, attached at its ends and forming a ring or spiral 8, through which a loop of the small intestine may slip.

3. A loop of intestines filled with contents may lie over a tense band of adhesions and thus become strangulated; this is a rare occurrence, but has been described by Treves.

4. The intestines may suddenly become kinked and occluded by traction from an adhesive band, as if an ovarian cyst were tapped and the sudden contraction drew on an adhesion to the intestines.

These bands and adhesions may occur between any of the viscera, the parietal peritoneum, omentum, and mesentery.

Adhesions may surround the bowel and contract, narrowing its lumen, also a mesenteric contraction may be a cause.

A coil may be caught between the pedicle of a tumor and the pelvic wall or may circumscribe a tumor pedicle.

Strangulation by Meckel's Diverticulum.—Meckel's diverticulum is due to the incomplete obliteration of the vitelline duct, and forms a finger-like projection from the ileum, usually within 18 inches from the ileocecal valve. As a rule, it is about 3 inches long, though frequently longer, and cylindric in shape with a conic end, though the latter is occasionally "clubbed." The end may become attached to the abdominal wall near the navel, to the mesentery, or to some other point, and thus form a band or loop under which strangulation may occur. More often the diverticulum is free and may form a ring into which its end projects. A loop of the intestines may enter the ring and, especially if the tip is club shaped, may push it before it and tie a knot (Fig. 230).

The vermiform appendix may become adherent to some point in the peritoneal cavity and form an arch under which a loop of the intestines may become strangulated. This may likewise occur with the Fallopian tube.

Strangulation of the Bowel Through Slits and Apertures.—This type is less often met with, and is, in fact, quite rare. Slits and apertures in the mesentery and omentum may be congenital, but are more frequently traumatic. They generally occur in the mesentery near the lower part of the ileum. Fissures, holes, or rings are more frequently formed by peritoneal adhesions; more rarely strangulation may occur in a tear of the uterus or bladder.

Strangulation from Internal Herniæ.—These are situated within the abdominal or thoracic cavity, or are subperitoneal or retroperitoneal, parallel to the abdominal wall without passing outward.

In some of the so-called external herniæ no swelling can be detected externally. Moreover, hernia may develop in an accessory form between the muscles and fascia.

Among internal herniæ we may have those of the recessus duodenojejunalis, intersigmoid recess, retroperitoneal, anterior, retrocecal, foramen of Winslow, and diaphragmatic.

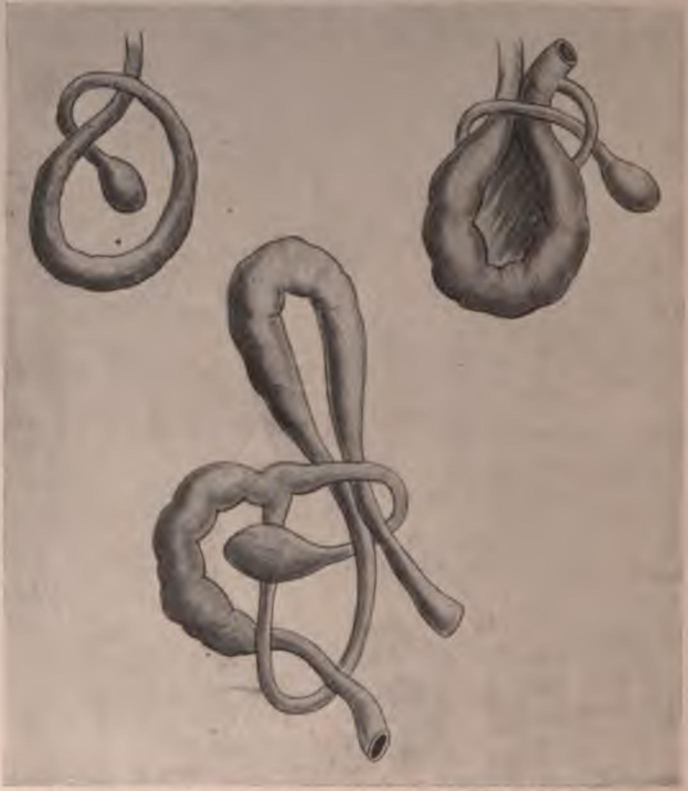

Fig. 230.—Knotting of a Meckel's diverticulum which has a button-like swelling of its extremity (after Treves).

Diaphragmatic hernia is most frequently met with of these forms and can occasionally be diagnosed.

It may be congenital or traumatic as from wounds, contusions, or excessive vomiting.

In the true form, a hernial sac of peritoneum or pleura covers the viscera; in the false form (the more frequent), it does not. This type is found on the left side, the stomach being involved most frequently, the colon and small intestine less so.

The stomach fundus passes into the thorax and apical rotation occurs.

Physical Signs of Diaphragmatic Hernia.—Pneumothorax, less motility of the side of thorax involved, bulging of the walls of the thorax, and metallic sounds are the chief symptoms. Dyspnea, distress or pain in the thorax, and difficulty in swallowing (dysphagia) may be present. Diaphragmatic hernia can be diagnosed by inflation with air or water, the use of the *x*-ray, or gastrodiaphany.

Mechanism of Internal Herniaform Strangulation (Compression of the Intestines).—The *mechanism* of the type of obstruction just described is as follows:

A coil of gut is drawn beneath a band or through an aperture, and becomes at once strangulated, or congestion of the mesenteric vessel occurs, gas and pus accumulate, and later strangulation occurs, or, in addition, a torsion of the bowel may take place—necrosis, gangrene, and perforation result.

The lower part of the ileum is chiefly affected. Peritonitis may occur early (in twenty-four hours) or late (in one to two weeks).

Sex and Occurrence.—In males 70 per cent., though some claim occurrence in the sexes is equal. Between twenty and forty years cases chiefly occur, though 40 per cent. are between fifteen and thirty years. In 90 per cent. the seat of the trouble is in the small intestine, usually the ileum; in 67 per cent. strangulation is in the right iliac fossa; in 83 per cent. in the lower abdomen (Osler).

Clinical Symptoms.—These are characterized by their sudden acute onset in perfect health. Rarely injury, violent movement, or diarrhea may precede the attack, and are considered to be factors. Sudden severe pain, occasionally colicky, often severe throughout, though at the end it may lessen. The pain at times corresponds to the seat of the strangulation. Vomiting begins early and is persistent and soon becomes feculent; *absolute constipation*, no flatus. There is early and rapid appearance of collapse, urine is scanty, meteorism slight or absent, slight tenderness on pressure, no blood in bowel movements if such occur, nor is it discoverable by enema. Death occurs in two to four days.

If peritonitis occurs, meteorism is marked and pain recurs and is severe. A circumscribed area of dulness or tumor is rare. The attack is of a fulminating character.

Volvulus may be defined as an obstruction of the bowel caused by a rotation of the intestines about their axis, a rotation of the bowel about its mesentery, or an intertwining (rotation) or knotting of two intestinal loops with their mesenteries.

It occurs most frequently in the sigmoid flexure. The ascending colon, cecum, and small intestine, especially the ileum, may sometimes be affected, and intertwining of the intestines is most often met with in this location.

Mechanics.—The Sigmoid Flexure.—The sigmoid flexure must be large and its mesocolon long and narrow for the development of volvulus. The ends of the sigmoid are thus approximated, and it can readily rotate around the mesocolon as a pedicle. This condition may be congenital or due to some chronic inflammation.

Etiology.—Chronic habitual constipation is the *chief cause*, as the weight of the fecal matter produces local displacement and distorts and elongates the mesentery.

The rotation is usually the result of bodily exertion. On the other hand, one branch of the flexure, being overdistended, may drop down over the other. The branches may be rotated through 180 to 360 degrees, or even several complete rotations may occur. The distention of the sigmoid, the congestion and exudation of fluid and accumulation of gas in the loop, prevent restitution to the normal position. The accumulation of gas in the colon also prevents return to normal.

Small Intestine.—The same conditions favor axial rotation of the small intestine.

Inflammation of the mesentery is sometimes a cause, also gallstones, by producing colic and spasm.

Accessory Causes.—Leanness, the absence of fat in the mesentery, and hence lessened intra-abdominal pressure have been considered predisposing causes, also trauma, jumping and lifting, sudden diarrhea (peristalsis) in these constipated cases, or a large enema (according to some).

Age.—It is more common in older than in young subjects, generally in those over forty years of age, though Fitz places it more frequently at thirty to forty years in his statistics.

Frequency.—Fitz finds 42 out of 295 cases of acute intestinal obstruction due to volvulus.

Sex.—According to Fitz it is more frequent in men—68 per cent. Some place the proportion much higher, as 3 or 4 men to 1 woman.

Symptoms.—This condition is characterized by its acuteness and rapid course. It is more rapid than any other form except, possibly, internal strangulation of the intestines.

Pain is sudden, violent, and never absent, and sometimes localized in the left lower quadrant; it may remit, but never a complete intermission. Vomiting is, as a rule, violent and profuse, continuous, and is an early symptom, though occasionally less frequent in sigmoid volvulus. *Fecal vomiting is comparatively rare. Constipation,* both for *feces* and *flatus*, occurs, as a rule, from the incipiency of the attack. Occasionally history of violent exercise or of acute diarrhea may precede the attack. Tenesmus may be present in volvulus of the sigmoid, though not as frequent as in intussusception, and in rare instances there is the passage of a small amount of blood.

Local meteorism is an important symptom, *the left lower portion* *of the abdomen* protrudes as a tense and elastic swelling, feeling like a rubber ball; visible peristaltic movements do not occur; percussion may be tympanitic, there may be metallic sounds or, rarely, dulness if much edema. In some cases there is an S-shaped protrusion of the abdomen, most prominent in the upper left and lower right quadrant. This is in the early stages. General meteorism develops most rapidly, *next in rapidity to general peritonitis*, but in the latter the abdomen is very tender; while in the former it is only slightly so. Within forty-eight hours the abdomen may be balloon shaped. This *rapid meteorism is an aid to diagnosis*.

The symptoms are very acute and collapse is marked. In axial rotation of the small intestine at the onset there may not be absolute constipation; the local meteorism starts higher up and there is no tenesmus. In these cases, curiously enough, the symptoms are no more violent than in sigmoid volvulus.

Anatomy.—There is local meteorism, the walls of the distended loop are thickened, rigid, edematous, and dark red. The peritoneal coat is often torn, as is the muscularis. Hemorrhages are seen in the intestinal wall. Blood, gas, mucus, and feces lie in the loop. The mesentery is hyperemic and infiltrated. The twisted part of the intestines is attenuated and pale. Gangrene may occur at the line of demarcation. Other parts of the intestines are collapsed and pale unless general meteorism subsequently occurs.

Course.—The course of volvulus is always acute. The patients may occasionally die in twelve to twenty-four hours or in two to three days; the average course is a week. They die of collapse, exhaustion, peritonitis, or from paralysis of the heart due to compression from tympanites. Unless operation is performed the cases, as a rule, are fatal, especially those with complete axial rotation.

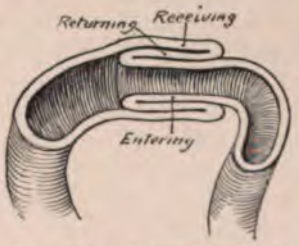

Fig. 231.—Intussusception.

Some patients who, from symptoms, seem to have suffered from partial rotation, the course being *more* chronic, have recovered.

Intussusception; Invagination.—It is in this condition that one segment of the intestines slips into an adjacent segment, so that the latter forms a sheath for the former. The two portions make a cylindric tumor which varies in length from an inch to many feet. The condition is always a descending intussusception. The outer tube is called the sheath or intussuscipiens; the middle and inner tubes, the intussusceptum; the innermost tube, the entering tube; the middle one, the returning tube (Fig. 231). The upper part, where the middle tube bends over into the sheath, is called the neck; its lower part, where the inner tube bends over into the returning cylinder, is called the head. Mucous membrane is in contact with mucous membrane and peritoneum with peritoneum. The one depicted is the usual form; sometimes a double or triple invagination may occur, or an incomplete lateral invagination; the reverse of a diverticulum. The mesentery participates in the invagination and becomes compressed and wedged in the sheath. The serous surfaces may become adherent, so that the invagination cannot be disengaged. There are various types of intussusception which may occur in every portion of the bowel:

1. *The Ileocecal Form.*—In this type the ileocecal valve forms the apex of the intussusception, the ileum the internal cylinder, and the colon the sheath; this often forms a very long intussusception, so that the valve may even protrude at the anus.

2. *The Ileocolic Form.*—The lowest part of the ileum forms the apex and protrudes into a cecum. If the cylinders become adherent, then the ileocecal valve and cecum may become inverted.

3. *Ileaca-ileocolic Form.*—There is first an ileac invagination which may force the intussuscepted part into the colon.

There may also be intussusception of the ileum, jejunum and ileum, jejunum, duodenum and jejunum, duodenum, colon, colon and rectum, and, rarely, of the rectum.

The ileocecal form seems to predominate, and is especially frequent in children.

The ileocolic is also quite frequent, and these two types give the longest intussusceptions.

In adults the ileac and ileocecal forms occur with about equal frequency, and in the colon it is quite frequent.

Intussusception of the vermiform appendix into the cecum has been reported, and also of the ileum into the open Meckel's diverticulum, when this is patent at the umbilicus, and also by the gut, grasping it or by traction.

The mesentery exerts traction on the intussusception and the bowel curves so that the concavity points toward the mesenteric attachment. This may cause a kinking, which further closes the intestinal lumen. Circulatory changes take place in the intussusception, edema, swelling, etc., and in severe cases strangulation and gangrene. The intussusception may slough off. Peritonitis is first noted on the second or third day; it may be local or become general.

Mechanism of Intussusception.—The probable reason of intussusception being so frequent in young children is that during the early months of the infant's life there is a rapidly increasing disproportion between the transverse diameters of the large and small intestines, the large intestine increasing very rapidly in diameter, so that the ileum can readily prolapse. For the production of invagination there is probably a local spasm of a portion of the intestines and the normal gut below is pulled upward by its longitudinal fibers over the contracted piece of bowel, and the irritation caused by the invaginated part then causes spasmodic contraction of the gut above, which carries the incarceration further downward.

Numerous experiments have been carried out for a study of the mechanism. None of these seem to show that primary paralysis is a factor, but that the condition is rather of a spasmodic type.

Paralysis of a limited part of the bowel may, I believe, be a factor in some cases.

Frequency.—Weiss finds that out of 321 cases—

	Per cent.	Per cent.	Per cent.	Per cent.
In the newborn and sucklings	Iliac, 24	Ileocecal, 42	Ileocolic, 10	Colic, 24
Childhood to puberty	Iliac, 23	Ileocecal, 43	Ileocolic, 14	Colic, 26
Adult	Iliac, 29.5	Ileocecal, 34.5	Ileocolic, 4.5	Colic, 27
Meckel's diverticulum (adult).	4.5			

177 in first year; 85, two to fourteen years; 59, later age.

The agonal type of intussusception occurs just before death, is of no importance, and is often multiple. Of Leichtenstern's cases, 131 out of 543 occurred in first year, 80 of them in four to six months.

Age.—Most frequent in infancy and early childhood, up to the fifth year.

Causes.—Diarrhea, intestinal polypi, carcinoma and stricture, ingestion of irritating food, contusion of the abdomen, shaking the body, and acute and chronic diseases have been given as factors.

Benign tumors when present, such as fibroma, myoma, especially the polypoid form, are generally at the apex of the intussusception, and most frequently occur in the ileum. They may set up peristalsis, and constriction and invagination so result.

It is rare with carcinoma or stenosis, and it may occur when the tumor is pedunculated. Often the cause is not discoverable.

Symptoms.—In the acute cases the attack is characterized by its suddenness. There may be preliminary intestinal disturbances, such as diarrhea or colicky pains. Often they appear while the patient is quiet, asleep or nursing. I shall refer to the acute cases only in this chapter. The iliac and ileocecal forms are the most acute. The colic and rectal form are more gradual.

Pain.—There is first pain of a violent colicky character, at times arising at a definite point. It may be very severe and overwhelm the patient, and in children may cause convulsions, or they may scream and groan. It is continuous at first, later may intermit, though at times it may be continuous throughout. In some cases the pain is in the right iliac region, or occasionally at the umbilicus, and at times local tenderness is present. Spasms and contraction with rigidity of loops of intestines sometimes occur in acute cases, but more usually in the chronic. Vomiting is constant and early in children, is not constant in adults, and hence a less important symptom in them.

The vomiting depends on the position of the intussusception; the lower down it is, the less likely the vomiting at the outset; peritonitis brings it on.

Feculent movements may occur at first; later, diarrhea with blood and mucus, and tenesmus if the invagination is low down. In some cases this is quite marked; *hemorrhages* from the *bowels ensue*, even if the invagination is high up.

Fecal vomiting is rare. Vomitus usually contains gastric contents, mucus, and bile, and only occurs in one-fourth of the cases in adults. The higher up the invagination, the sooner it appears. Meteorism of a great degree is exceptionally present; sometimes the abdomen is even retracted. It is dependent on the degree of constriction.

Tumor.—A palpable tumor can at times be appreciated in about one-half the cases. It varies in size, may seem shorter to the touch than it really is, or it may impart the feel of a double swelling; it is usually of a cylindric or sausage shape, elongated, and curved. Its consistency may vary at different times and it can be slightly compressed. It may feel hard and then suddenly soft, or even vanish. The changes in *consistency and resistance* are due to the spasmodic *contraction* in the intestinal wall. During contraction it may be tender.

Situation.—Most frequently the tumor is located in the region of the sigmoid; then at the anus or rectum, then at the cecum, descending transverse or ascending colon.

The position of the tumor may be permanent if there are any adhesions, or it may change if the intussusception increases.

Prolapse of the swelling through the anus often occurs.

Duration.—Especially in children severe symptoms of collapse may take place within two to three days, and if vomiting persists, it may be feculent.

About 80 per cent. of young subjects die; subacute cases run two to four weeks and spontaneous cures occur in this type.

Death from collapse, gangrene, and peritonitis may occur during the first week in infants, or in two to three weeks in adults. Sepsis and phlebitis may be associated.

Diagnosis.—Acute commencement, vomiting constant in children, but not so in adults; at times tenesmus, the *presence of bloody stools;* then retention of feces and flatus, *distention of the abdomen* usually slight; and last the appearance of a tumor are diagnostic of intussusception.

Obturation of the intestines signifies the occlusion of the bowels by a foreign body situated therein; among such are gall-stones, enteroliths, foreign bodies which have been swallowed or, rarely, inserted into the rectum, and masses of fecal matter.

Gall-stones, in exceptional cases if very large, may cause an acute intestinal obstruction. They probably have ulcerated through into the bowel from the gall-bladder. They may become impacted in the duodenum, jejunum, or at the ileocecal valve.

Cases of this last type are more frequent in women than in men. In many there is a previous history pointing to cholelithiasis. The symptoms are pain, violent vomiting (generally bilious), which frequently becomes feculent, constipation, which is not always absolute, meteorism, often not pronounced, and collapse. In some cases a hard resistant swelling may be felt. In others there will be some flatus from the rectum. Milder symptoms of occlusion may occur, evidently when the lumen of the bowel is not completely shut off. The patient in this class of cases would suffer from attacks of colicky pain, vomiting, and constipation, but no collapse. These cases may also have perforative peritonitis, and often die on the fifth to tenth day unless operated on. At times the stone may move onward and escape, with recovery of the patient.

Occasionally there will be some diarrhea with blood during the onward movement of the calculus, the result of traumatism to the mucosa.

Enteroliths.—An intestinal calculus rarely causes acute obstruction unless it has been dislodged from an intestinal diverticulum, and suddenly occludes the bowel. It generally produces the symptoms of chronic obstruction or digestive disturbances and obstinate constipation.

Calculi ordinarily consist of carbonates or phosphates with a foreign body as a nucleus; or as a result of the prolonged use of drugs, such as chalk, bismuth, magnesia or salol, or some indigestible material, such as oat-stones from oatmeal, etc., in which inorganic salts are deposited. They frequently develop in the large intestine.

Foreign Bodies.—These are accidentally or intentionally swallowed. This is especially apt to occur with children or with insane or hysteric persons. Occlusion has also been produced by the insertion of a foreign body into the rectum. Among such substances are knives, spoons, forks, keys, marbles, stones, false teeth, fruit stones, neckties, hair or beads, pins and needles; in fact, all varieties of objects. In some cases the symptoms may first be chronic, when suddenly acute occlusion will occur; in others it is acute from its incipiency. Murphy's button has caused occlusion, also a large mass of tapeworms. Pedunculated tumors of the intestinal wall, such as polypi, fibroma, and myoma, may produce it.

Many of these objects can be readily recognized by means of the x-rays and the fluoroscope, or by a Röntgen picture.

Fecal masses may completely occlude the intestines as a result of habitual constipation. The symptoms are usually subacute or chronic at first, and finally become acute from complete occlusion.

Dynamic Ileus (Intestinal Paresis).—Acute obstruction produced by paralysis of the bowels is called paralytic or dynamic ileus. Among the various causes are hydrocele, contusion or inflammation of the testicles, etc., which may cause a reflex paresis; paracentesis of ascites; trauma over the abdomen; after laparotomy from manipulation of the viscera or following removal of a large tumor, or after Cesarean section; other operations from the effect of the anesthetic; damage to the intestines, as from a hernia after relief of the strangulation; local or general peritonitis; renal or biliary colic may cause reflex obstruction, possibly due to spasm or paresis; overdistention of the intestines from gas or feces; various toxemias, as from typhoid or pneumonia or from any acute infection, also sepsis, as from appendicitis, and as a concomitant of shock or uremia.

Congenital Causes.—Congenital occlusion of the rectum or colon produces acute obstruction.

Acute Obstruction Engrafted on Chronic Obstruction.—Chronic stenosis with chronic constipation may suddenly produce an acute obstruction.

Pathology of Acute Obstruction.—In acute obstruction the pathologic findings differ somewhat, according to the various types and factors. There has been an unfortunate tendency to confuse the findings in acute and chronic obstruction. If we have an acute condition engrafted on a chronic obstruction, we may then find above the point of obstruction dilatation and hypertrophy of the intestines, catarrh, and ulcerations.

In *true acute occlusion* the process is *too rapid* for these changes, but the following occur: the intestines below the occlusion are empty and contracted; above it the intestines are markedly dilated and distended and the walls are thinner. If a loop is involved, the walls are thick and congested (but not hypertrophied). The coils above the obstruction are distended, filled with gas and ill-smelling contents. If the small intestine is occluded the distention will involve the gastrointestinal tract above the occlusion; if the large intestine is occluded the colon will be distended first; later regurgitation through the ileocecal valve occurs and universal distention.

In a loop which is strangulated there is local meteorism and it may be markedly distended; there are congestion, edema, hemorrhage, a dark red color of the gut and gangrene, with general peritonitis and bloody fluid in the cavity; or a single perforation from ulceration or gangrene may occur; and rarely, if the case be prolonged, a local abscess or local peritonitis.

General Symptoms of Acute Obstruction.—As a rule these begin very acutely, though rarely there is a previous history of diarrhea or constipation, improper food, a laxative, traumatism, or of violent exertion.

There are at first violent abdominal pains, colicky in type, local or diffuse. The pain is continuous, though sometimes it may remit. Nausea, hiccough, and vomiting first of the gastric contents, later bilious, and, finally, feculent vomiting rapidly follow. Meteorism quickly appears; there is absolute constipation and no passage of flatus. Tympanites increases, a great increase of intra-abdominal pressure is present, and the muscles become tense and the entire abdomen tympanitic; respiration is markedly interfered with, the breathing is rapid and shallow, pulse rapid and feeble, extremities cold, cold sweat, face pale, eyes sunken, and extreme thirst; total collapse and the patient rapidly succumbs.

Only a brief analysis will be made of these symptoms, as they have been thoroughly described under the respective causes of acute obstruction. Pain is the most constant symptom and it *never remits* completely. As a rule, it is more acute in obstruction of the small intestine; the initial pain is probably due to irritation of the intestines and peritoneum, later to spasmodic intestinal contraction, and finally to peritonitis. In the last stage pain may cease as a result of the terminal fatal intestinal paresis. External pressure, as a rule, increases the pain.

Active and visible *peristaltic movements* and tetanic stiffening of the bowel are *exceptionally* seen in acute occlusion, when the intestines were previously healthy and unobstructed; and if they are visible, are never as marked as in cases of acute obstruction *supervening on a chronic stenosis of the intestine*. This is an important point to remember. The visible peristaltic movements if present, in connection with the other symptoms, are an aid to diagnosis.

Vomiting is nearly always present. At first it is probably due to reflex irritation of the nervous system; later it has been ascribed to antiperistalsis; or to mechanical causes, as explained by Haguenot, who states that fluid contents accumulate above the obstruction; and from gas pressure and contraction of the abdominal muscles the contents are forced into the areas of less resistance and thus reach the stomach, where they cause vomiting.

Feculent vomiting often occurs when the obstruction is in the small intestine, due to putrefactive processes therein.

Constipation is usually marked; though rarely there is diarrhea with blood, as in intussusception.

Absence of flatus is quite significant.

Meteorism.—This may first be circumscribed (local), there being tympanites and protrusion of the intestines for a short distance above the point of obstruction; thus, a protrusion of the right side would first be noted if an obstruction occurs at the hepatic flexure. Later the meteorism becomes general and the *abdomen barrel shaped.*

Collapse.—This is marked, especially so if there is strangulation, and it is more rapid if the small intestine is involved.

Thirst and dryness of the tongue are present, being due to vomiting, sweating, and increased intestinal secretion, which diminish the fluidity of the blood.

Coma, delirium, and fever rarely occur.

Diagnosis of Acute Intestinal Obstruction.—Having described the symptoms of acute obstruction, a careful study of the physical igns and the methods of physical examination are necessary to complete the diagnosis.

Investigation of the type and degree of meteorism (tympanites), both by inspection and percussion, is of great importance. At this point I again desire to call to my reader's attention that acute dilatation of the stomach presents many symptoms of ileus and may obscure the diagnosis. The *pain* of acute ectasia is not as severe and continuous as with acute intestinal obstruction, in *that it is immediately relieved by lavage.* The stomach may occupy the entire abdominal cavity, though usually it fills the left half and lower part of the abdomen. The vomiting of acute ectasy is also peculiar, in that it is very profuse, incessant, and in large amount, and comes up in gulps without straining. It is usually watery and greenish in hue, though it may be brownish or black. If the stomach alone is involved, distention will disappear after lavage.

Inspection.—If there be occlusion of the jejunum or duodenum, unless the stomach also be greatly dilated, the upper part of the abdomen is protruded slightly.

Occlusion at the cecum or lower ileum gives the so-called "ladder" pattern, the coils lying one above another either obliquely or transversely in the abdomen, and the distention is more central.

In stenosis of the sigmoid flexure the upper and lateral aspects of the abdomen are usually distended, and we have so-called flank meteorism.

When the distention is limited to a section of the colon, flank meteorism may be unilateral; thus the right iliac region is intensely tympanitic if the occlusion involves the hepatic flexure. If the small intestine alone be included, the distended loops are seen in the center and flank meteorism is absent.

With volvulus the left lower portion of the abdomen protrudes; or an S-shaped protrusion of the abdomen, most prominent at the upper left and lower right quadrants, occurs.

We must remember that local meteorism occurs only early, and later general tympanites ensues, so that we may not see the case early enough to avail ourselves of these data.

Peristaltic waves, if present, aid to locate the obstruction.

Palpation.—In some cases palpation reveals a circumscribed area which is tender on pressure, and may aid in localizing the obstruction. In others a tumor is palpable, especially with intussusception, occlusion by tumors, or fecal impaction.

Palpation of the hernial openings is necessary. Digital examination of the rectum and vagina are most important. We may feel a stricture or intussusception in the rectum and bloody fluid may escape. I deprecate the method of inserting the hand in the rectum.

Percussion.—Local meteorism gives a deep and loud note which is not truly tympanitic, but often of a metallic ring, and helps locate the obstruction. Exceptionally dull percussion may be heard over the swelling, due to edema of the intestinal walls or to accumulation of fecal contents or blood admixture.

In normal subjects the percussion note in the upper lumbar region behind is high, flat, and dull. It is loud and deep in stenosis of the large intestine. When the obstruction is in the sigmoid, the loud and deep note is found on both sides, and when in the splenic flexure or transverse colon, then only in the upper lumbar region of the right side.

If there is general tympanites and no change of percussion is noted for a long time over any one region of the abdomen, there is probably intestinal paresis. General tympanites, with absence or diminution of liver dulness, does not always mean perforation of the bowel, though it frequently does so. One can often determine peritoneal exudation by percussion.

Auscultation.—Splashing and gurgling noises often demonstrate that peristalsis is marked. Succussion sounds and fluctuation on palpation are frequently found in the intestines above the point of stenosis. Examination of the vomited matter will show whether fecal material is present. Urine is scanty and albuminous, and often shows indican and gives Rosenbach's reaction, especially in obstruction of the small intestine. Suppression of urine occurs in this condition.

Differential Diagnosis Between Obstruction of the Small and Large Intestine.—*Obstruction of Small Intestine.*—Pain, vomiting, and collapse are more acute and appear early. Early feculent vomiting. *Indicanuria early, on second or third day. Anuria early, early meteorism, and often is high up.* Absence of early indicanuria (excluding peritonitis and intestinal inflammation) tends to show obstruction is not in the small intestine.

Vomiting in duodenal obstruction is rarely feculent, but is so if obstruction is lower down.

Obstruction of Large Intestine.—If the disease is chronic or runs a long course, absence of indican shows lesion is present in the large intestine; its presence, however, tells nothing, as it may appear late.

General symptoms are usually less violent, though they are violent in volvulus. Fecal vomiting occurs later. Meteorism is lower down in the early stage of acute obstruction.

Tenesmus and *blood* in the stool suggest an obstruction low down. Brinton's method by the injection of water is sometimes of service. If not more than 1 quart can be injected, the obstruction is probably low down; if 2 quarts, it is probably above the sigmoid, and if 4 quarts, then in the commencement of the colon or higher up.

Inflating the bowel with CO_2 by Rose's bottle or by air will fill the large intestine up to the obstruction. This method is of no service if there is very marked distention, and lavage should be first performed to reduce it.

Differential Diagnosis Between the Different Forms of Acute Obstruction.—This is often difficult. In some cases it is possible, but in others we can only conjecture the probable type.

Acute strangulation in herniæ, omental slits, or by bands constitutes about 34 per cent. of cases. This is more frequent in males between twenty and forty years. In 90 per cent. the small intestine is the seat of trouble, mostly in the ileum and lower abdomen or right iliac fossa. The symptoms are sudden and acute. There may have been a previous history of peritonitis, hernia, or injury. Pains are severe, vomiting begins early and soon becomes feculent, absolute constipation and no passage of flatus. Tenesmus absent. Collapse is early and marked. Urine is scanty and meteorism slight. Attack is of fulminating type. Physical examination often gives no definite data.

Volvulus.—This most frequently involves the sigmoid. History frequently of chronic constipation is more common in males and in those over forty years of age, from forty to sixty, though Fitz's statistics show frequency from thirty to forty years of age. If volvulus of small intestine, it cannot be differentiated from incarceration. Pain is sudden and violent, it may remit, but never intermits. Vomiting is quite common, but may be absent at first in volvulus of the sigmoid, and in the latter case fecal vomiting may not be present. It occurs in some cases, but is *not as frequent* as in internal strangulation; in fact, it is rather rare. Meteorism occurs early and is at first *local* in the *lower left quadrant*, or as an S-shaped protrusion. General meteorism rapidly occurs, and thus is an aid to diagnosis. The sigmoid can occasionally be felt *as a tumor.* Constipation and absence of flatus are usually complete; occasionally there is a little blood in the stool and only a moderate amount of water can be injected into the rectum. Symptoms are acute and collapse is marked.

Intussusception.—Most frequent in infancy and early childhood. Onset is sudden. Pains appear early, are colicky in character; in children, may cause them to scream or have convulsions; pains are paroxysmal. Vomiting occurs *early* and *constantly* in children; a less important symptom in adults.

Invaginated coil can be appreciated or a tumor in about one-half the cases. Occasionally the swelling may prolapse through the anus.

Tenesmus and *evacuation of blood* occur, then constipation and retention of flatus. Fecal vomiting rare, unless case is prolonged; collapse is early in children (in two to three days), but not as early as in strangulation; collapse is slower in adults.

Obstruction by Gall-stones, Enteroliths, and Foreign Bodies.—Occlusion from gall-stones occurs chiefly in older women. There is at times a previous history of gall-stones, jaundice, and liver enlargement, or tenderness, which aids our diagnosis. The obstruction usually occurs in the small intestine. The general symptoms are often not so severe as in other types, there being some flatus passed at times and in some cases slight diarrhea with blood. If obstruction of the ileum, feculent vomiting is more marked. Meteorism is not so marked and collapse not so great. Occasionally the stone can be palpated.

Enteroliths.—Their recognition is quite difficult unless small fragments have been voided. They develop most frequently in the large intestine. Symptoms of chronic obstruction or digestive disturbances and obstinate constipation are more frequent. Acute symptoms are more rare.

Foreign Bodies.—The previous history and use of the x-rays will determine these. Obstruction from accumulation of pits will generally be suspected by reason of the appearance of some of them in the stool.

Fecal Accumulation.—This rarely gives the picture of acute obstruction unless a tumor or stricture of the intestine be present, when accumulation may suddenly cause complete occlusion. Rectal examination usually shows hardened scybalæ and palpation demonstrates a hardened mass in the colon (descending sigmoid or cecum especially). If the mass be softer, it will impart a doughy feel on pressure.

Dynamic Ileus.—Acute gastroduodenal dilatation of the stomach presents symptoms of intestinal obstruction, and commences with acute pain, undoubtedly due to stenosis of the duodenum by pressure. Under Diagnosis of Acute Obstruction I have described the peculiar type of vomiting in acute ectasy and also the position of the stomach. The pain, distention, and other symptoms are relieved by frequent lavage and by the adoption of the abdominal position (the patient lying on his belly).

Obstruction of the bowel from a paralytic condition involves, as a rule, a considerable segment, or frequently the entire tract. After reduction of a hernia probably only a small segment is first involved, but I believe paralysis of a considerable segment, and in many cases of the entire bowel, finally results. The acute cramp-like pains, *persistent and paroxysmal*, are absent in this type of obstruction (really not a true obstruction at all), which always occur in the early stages of other types of obstruction. When acute spasmodic pain *is present and persistent*, I always feel positive of a true obstruction. The vomiting, constipation, collapse, etc., may be similar to obstructive ileus. There may be sudden pain with the acute distention, so that perforation is suspected, but after lavage and enteroclysis the pain disappears, and muscular rigidity is found to be absent. These features exclude, first, "obstructive ileus," on account of disappearance of the pain; and second, "peritonitis." In the early stages of "dynamic ileus," distention, interference with the cardiorespiratory functions, and obstinate constipation precede the vomiting and severe syptoms as a rule. There is usually more of a sense of painful discomfort and oppression, except in the cases of sudden acute distention.

In the terminal stages of true obstruction, paresis of the bowel may occur; and then *spasmodic pain disappears;* in paresis of the bowel from peritonitis there is the general pain and tenderness from the peritonitis, but not the acute spasmodic pain peculiar to true obstruction.

In true dynamic ileus, therefore, there is the absence of that acute *persistent spasmodic* pain of severe type which is present in all cases of true obstruction.

Differential Diagnosis Between Intestinal Obstruction and Other Diseases.—*Acute Peritonitis.*—Generally a history of local peritonitis, such as appendicitis with pain localizing in right iliac fossa, and then sudden cessation of the cramp-like pains, followed by abdominal tenderness, muscular rigidity, great distention, and frequently rise of temperature. Vomiting generally begins later and feculent vomiting still later, etc.

In obstruction the character of pain is more severe and persistent, as a rule, and often in the umbilical region or in the left lower quadrant; temperature is subnormal at the start; abdominal tenderness is not as marked; feculent vomiting earlier; often local meteorism before general tympanites. General pains are colicky and persistent until peritonitis and paresis set in. The *paralytic form of ileus* may often occur with infectious diseases, or acute appendicitis with peritonitis (general), also after operation or inflammation in the pelvis or genito-urinary organs; and the knowledge of these facts aid our diagnosis. We must remember that in hysteric women all the symptoms of ileus, even to fecal vomiting, may occur without there being any obstruction.

With biliary and renal colic there may be a reflex paralytic ileus, but the symptoms of these conditions aid the diagnosis.

Lead- and arsenic-poisoning have sometimes been mistaken for ileus, but again we have the history and other symptoms, especially of enteritis. Simple intestinal colic soon subsides under treatment. Acute pancreatitis and enteritis give their symptoms.

There is tenderness in the course of the pancreas if this is involved, a circumscribed epigastric swelling, and tender spots throughout the abdomen (Fitz); but in some cases it is difficult to differentiate pancreatitis from obstruction.

Course.—The course of acute obstruction depends upon its cause and site. The higher up in the intestine, the more acute the course, as a rule.

Volvulus and strangulation are very acute. The patient may die in collapse within a few hours, or the course may be prolonged to two to three days or even a week. Intussusception, if unoperated, may last several weeks. If the patient survives the collapse, and the patency of the gut becomes reëstablished (as in intussusception, foreign bodies, or volvulus), flatus is first passed and then later a fecal movement, and the symptoms gradually abate. The invaginated bowel has been known to slough off and be passed in the stool. From changes in the gut, due to ulcers, adhesions, etc., the patient may subsequently develop symptoms of chronic obstruction.

If operation is not performed, the patient usually dies of acute shock or peritonitis. The latter may be due to perforation or to direct penetration of the paralyzed wall of the gut by intestinal bacteria.

In some there may be a circumscribed peritonitis. With the diffuse peritonitis we have the symptoms already described.

Embolic processes may develop in the liver, lungs, and other organs. There may be aspiration pneumonia or, exceptionally, local abscess, with perforation of the abdominal wall, or into another part of the intestines or into other viscera, as into the stomach, bladder, vagina, or uterus.

Prognosis.—The prognosis of acute obstruction is very serious. Some observers state that about one-third of all cases recover, however.

Obstruction caused by coprostasis, gall-stones, or some foreign body give the best prognosis. Dynamic ileus with modern methods of treatment, I believe, is next in regard to favorable results, then intussusception, and the worst cases are volvulus and strangulation. The *earlier the operation, the better the prognosis.* The last types I believe fatal nearly invariably, unless early operation is carried out.

Treatment.—This may be divided into medical and surgical, and the respective indications are extremely clear. I shall first briefly classify these, giving the treatment in tabulated form.

Cases for Medical Treatment.—1. Acute obstruction due to fecal accumulation. Acute attacks are rare; they are, rather, *subacute* or acute engrafted on chronic:

(*a*) Lavage is indicated to relieve tympanites, if present, or if vomiting.

(*b*) Digital examination of the rectum and removal of scybalæ with the fingers, and then frequent enemata of soapsuds, olive oil, glycerin, or ℥vj (200 cc.) of magnesium sulphate (saturated solution) in water, 1 pint (500 cc.), followed by recurrent enteroclysis with normal saline solution at 110° to 120° F., using 2 to 3 gallons at a sitting, about ½ to 1 pint (250–500 cc.) being kept in the bowel. For the first twenty-four to forty-eight hours the treatment consists in the simple mechanical emptying of the rectum and large intestine. The enemata should contain about 1 to 2 quarts (liters), in which is olive oil, 1 pint (500 cc.), alone or with glycerin, ℥iv (125 cc.). It is well to give the enema with the patient in the knee-chest position, the buttocks elevated as high as possible. If vomiting or distention, *no food at all until this ceases;* thirst may be relieved by moistening the mouth, sucking a piece of lemon or orange, and, if necessary, by hypodermoclysis, rectal saline enemata, or proctoclysis. Milk is objectionable, as it forms curds and helps fill the bowels. Broths and soups should be given until the accumulation has been removed.[1] Strychnin by hypodermic, gr. $\frac{1}{60}$ to $\frac{1}{30}$ (0.00108–0.0021) three or four times a day, can be given as a stimulant. It also helps tone up the bowel. Tincture of belladonna in large doses, administered up to physiologic symptoms, ♏x (0.59) three or four times a day, is of service, as soon as it can be retained. Later, olive oil, ℥ij to iv (60.0–125.0), can be given several times a day by mouth. On the second or third day, if matters are progressing favorably, give cathartics, calomel, castor oil, etc., by mouth. Massage and external electricity are of value in these cases after the acute symptoms have subsided. I have frequently employed electric enteroclysis. I have seen it take several weeks to completely empty the bowel.

2. The second class of cases in which medical treatment is indicated is in dynamic ileus. The indications are:

(*a*) Abolition of food.

(*b*) Relief of thirst, as previously indicated.

(*c*) Frequent lavage to relieve intra-abdominal tension and also vomiting.

(*d*) Frequent enteroclysis (recurrent) with hot normal saline solution at 115° to 120° F., several gallons being used, a small part being kept in the intestines (preferably). High enemata with soapsuds, olive oil, and glycerin are also of service.

(*e*) Electric enteroclysis.

(*f*) Tincture of belladonna, ♏x (0.59), given three or four times a day up to physiologic symptoms. Strychnin, gr. $\frac{1}{30}$ (0.002), every three hours.

(*g*) Magnesium sulphate (saturated solution) by rectum, ℥iv (125.0). Heat locally is of value, and in one case the continuous local application of the ice-bag stimulated the bowels to contract. I prefer the ice-bag in many cases, unless collapse is present.

Cathartics by mouth immediately after lavage, especially calomel, gr. v (0.3), crushed up in water and poured in through the stomach-tube at the end of the washing. Plain water and *no saline solution* should be employed for lavage when *calomel is administered.* Physostigmin sulphate, gr. $\frac{1}{100}$ to $\frac{1}{60}$ (0.0006–0.001), every two to three hours for three doses. If all methods fail and the patient is rapidly losing ground, then simple enterostomy of the most distended loop, under cocain, as suggested by Elsberg in obstructive ileus, I believe, is indicated. This procedure allows the escape of gas and some contraction of the intestines to take place. Magnesium sulphate solution should be injected through the opening, and the procedures already suggested should be continued. Drainage should be closed after twenty-four hours. In my own experience I have had *good results from continuous and active medical treatment.* The lavage followed by catharsis by mouth is of *equal importance* as the *enteroclysis.* Proctoclysis is not sufficiently active.

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[1] This only refers to after the *subsidence of acute symptoms* and after the *bowels have acted.* No food is given before this time.

Obstruction by Foreign Bodies.—If the obstruction is by accumulation of fruit stones, an enterolith, or a gall-stone, and the case is seen on the first day of attack, lavage and the administration of ℥iv (125 cc.) of olive oil through the stomach-tube aids to lubricate its passage, followed by enteroclysis. *No cathartic* should be given by mouth. If the mass begins to come away and symptoms are relieved at once, then delay; otherwise, operate.

If the case is seen later, with progressive symptoms, use lavage and enteroclysis to relieve distention and operate at once.

Intussusception.—With infants or young children the stomach should first be washed out to relieve abdominal tension. An anesthetic is then administered and the child placed in the Trendelenburg position, and an attempt at reduction may then be made by inflation. A bellows is attached to a catheter, and the air should be injected slowly, the buttocks being held together. The best guide to the amount introduced is the tension of the abdominal walls; if tension is marked some air is allowed to escape.

This procedure should not occupy over fifteen to twenty minutes.

A saline solution, milk and water, or thin gruel at a temperature of 100° to 105° F., for the relaxing effect can be employed instead (as suggested by Holt). The fluid is suspended in a fountain syringe 4 or 5 feet above the patient's head, the tension of the abdomen being watched. Otherwise the procedure is the same.

Reduction is indicated by a rumbling sound and by the abdomen resuming its natural contour, with disappearance of the tumor; in some cases a gush of feces follows.

If these symptoms are absent, the abdomen is examined while the patient is still under chloroform, especially the right iliac fossa, for the continued presence of the tumor.

It is better not to repeat the injection.

If the tumor is present, or if vomiting continues and no gas or feces are expelled, or the pulse and temperature rise, immediate operation is indicated.

This method of taxis should be tried on the young in an early stage (the first day); if later, operate. If, on the other hand, there is immediate improvement, small doses of opium are given for a few days to prevent re-invagination.

Surgery.—In all other cases of acute intestinal obstruction except those noted, early, *preferably immediate, operation* is indicated.

A large percentage of fatalities imputed to operation are due to delay on the part of the physician; thus, in strangulation, volvulus, intussusception, and obstruction from foreign bodies (except under the condition of the rapid passage of foreign bodies, as noted above), immediate operation is indicated. *The earlier the operation* the better the prognosis.

In these cases cathartics should never be given, the bowel should not be massaged, nor electricity used.

Puncture of the bowel through the abdominal wall to relieve gas is absolutely dangerous—an invitation to peritonitis.

Laxatives do positive harm. The attempted diagnosis of the seat of the acute obstruction by the administration of bismuth by mouth and the use of x-rays, which necessitate a *delay of twenty-four hours*, should never be undertaken.[1]

Lavage (frequent) is of first importance; it lessens intra-abdominal tension and also the pain, makes diagnosis easier, and in some cases has actually proved curative. This last is explained by the fact that the gastro-intestinal distention is relieved above the point of obstruction, and occasionally the gut escapes from the constriction.

If the patient is seen on the first day of the attack lavage should be given at least twice, one or two hours apart, while preparing for operation; if later, then once before operation.

Enteroclysis at 115° to 120° F. is of value to lessen the distention due to general paresis and improve the pulse; if the intussusception lies in the rectum, the latter is contra-indicated.

No fluid or ice by mouth, but the tongue can be moistened and small normal saline injections or proctoclysis be administered for thirst. These methods also relieve postoperative thirst. Heat or cold to abdomen. Hypodermoclysis and infusion if there is shock.

Lavage often relieves the pain, but if this is very severe, morphin, gr. ¼ (0.016), by hypodermic, and repeat to lessen shock. All these methods are of use while preparing for immediate operation.

The general medical methods of opium or morphin for three or four days and expectant treatment, with *subsequent operative* fatality, *are to be deplored.*

Operate in acute peritonitis. Subseqeunt to operation, lavage, if vomiting, and nutritive enemata; no food or water by mouth; later, a little hot water. Open bowels by enema or enteroclysis in twenty-four to forty-eight hours, as intestinal paresis is usually present; earlier if the *symptoms persist* which are due to this condition.

Rectal injection of 400 cc. (℥xij) of 7 to 8 per cent. salt solution in intussusception to produce reversed peristalsis has been suggested by Riegel. Experimentally it has proved efficacious, but practically I am dubious of its value, as most cases of intussusception are adherent or strangulated.

In desperate cases enterostomy with drainage of the intestines, done under local anethesia (cocain), is advisable. A few hours later (within twenty-four hours) radical operation with relief of the obstruction can be performed. Elsberg[2] holds that preliminary enterostomy, leaving the prolonged search for the obstruction for a second operation, to be more frequently advisable.

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[1] Bismuth enema, followed by the x-rays, is only of use in determining obstruction in the large intestine. It is of value in chronic cases, but not advisable in acute ones.

[2] The Value of Enterostomy and Conservative Operative Methods in the Surgical Treatment of Acute Intestiual Obstruction, Annals of Surgery, May, 1908.

CHRONIC INTESTINAL OBSTRUCTION

In this condition there is a stenosis or narrowing of the lumen of the intestines, but the obstruction is not acute and complete in the earlier stages, but comes on gradually.

Etiology.—It may be caused by the same factors which produce acute obstruction, if the entire lumen of the canal is not occluded.

One of the most frequent causes of chronic obstruction is stricture resulting from ulcers or new growths. The latter, even if they do not occupy the entire canal, may protrude at one point and partially occlude the intestines. They may be benign or malignant.

In addition we have the peculiar tumor-like tuberculosis of the cecum, which causes a progressive stenosis, and chronic peridiverticulitis (sigmoiditis) may produce a narrowing of the lumen with symptoms. Both of the latter conditions often simulate carcinoma.

Strictures resulting from ulcers involve the large intestine much more frequently than the small, probably in a ratio of 6 to 1, according to Treves.

Among the causes of stricture are tubercular, stercoral, syphilitic, typhoid, dysenteric, and duodenal ulceration.

Woodward[1] has demonstrated that dysenteric ulcers rarely cause intestinal stricture, and Nothnagel agrees with him. Stercoral and tubercular ulcers are a quite frequent cause, as is also syphilitic ulceration. Typhoid ulcers are a rare cause.

There are other rare factors reported, such as ulceration in a portion of the bowel that has been incarcerated, or stricture following the sloughing off of invaginated intestines. A few cases of traumatism with damage to the intestines and subsequent stricture, or of a circumscribed peritonitis following trauma, and subsequent stenosis have been reported. Carcinoma of the pancreas, enlargement of the retroperitoneal glands, and gall-stones may cause stricture. A stricture may be congenital.

Rectal strictures are quite frequent, and much more so in women, as from syphilitic, tubercular, stercoral, and hemorrhoidal ulcers. Operation for prolapse of rectum or for hemorrhoids, especially the Whitehead operation, traumatism from the syringe-tip, or the introduction of foreign bodies may produce stricture.

Traumatism from the child's head during parturition may be a cause.

Gonorrheal abscess of the Bartholin glands may lead to ulceration of the rectum and stricture ultimately result.

Anatomy of Chronic Stenosis of the Intestines.—The intestines below the stenotic area are empty and contracted and the bowel normal. Above the point of stenosis the bowel is dilated, often to a great degree, and may form a sac-like pouch. The dilatation may involve only a short part of the bowel above the stenosis.

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[1] Medical and Surgical History of the War of the Rebellion.

In some cases there is considerable distention from gas and the abdomen may assume *the barrel shape.* The degree of distention depends on the tightness of the stricture. There is stagnation of the intestinal contents above the stricture, which causes mechanical distention and may stimulate the peristaltic action. This material also acts as an irritant. When the musculature is stimulated to increased activity *hypertrophy* is thus produced. This accounts for the violent visible peristaltic movements in chronic obstruction.

Patel claims that in stenosis from external pressure we have dilatation without hypertrophy, and that the latter only occurs if ulcers, which cause contraction through irritation, are present. This would explain the occurrence of hypertrophy without stenosis in some cases of intestinal ulceration.

Changes in the mucosa and submucosa are frequently present, a catarrhal condition and ulceration (stercoral). General peritonitis, local peritonitis, or local abscess may result.

The intestines may become elongated above the stenosis.

Location of Stenosis.—The large intestine is the most common seat of the stricture. Syphilitic, dysenteric, and stercoral strictures are chiefly localized here, as are also those resulting from traumatism or from follicular ulceration.

Tuberculous ulceration produces stricture most commonly in the small intestine, though tuberculous tumor is found in the cecum, and probably tuberculous ulcer is more frequent in the rectum than has been usually credited. Tuberculosis of the sigmoid also occurs.

Malignant strictures are most frequent in the *large intestine.*

If the ulcer producing the stricture lies parallel to the longitudinal axis of the bowel, stenosis is not as marked as when it is an annular ulcer (girdle ulcer). The stricture, as a rule, is short and the external aspect of the intestine looks as if a ribbon had been tied about it. The external surface of the gut is often covered with exudate, so that the bowel is thickened and there may be adhesions between the intestines and other loops or other viscera, which further constrict the intestines.

The stricture consists of cicatricial tissue unless malignant. Folds of mucous membrane near the cicatrix or hypertrophic polypoid protrusion of mucous membrane may aid in closing the gut.

If there are numerous stenoses, as from tubercular ulcers, there may be sacculated dilatation of the small intestine between the stenosed points.

Symptoms.—These depend upon the cause of the obstruction: malignant growth must be differentiated clinically from benign conditions. The symptoms generally come on gradually.

In stenosis of the small intestine they may be latent for a considerable time, on account of the fluidity of the bowel contents, and then appear with rapidity. On the other hand, stenosis of the colon produces symptoms more rapidly on account of the solid contents.

As a rule, *constipation* is one of the earliest symptoms, and this gradually becomes worse. The patient complains of digestive disturbances and swelling of the abdomen, there is loss of appetite, and nausea. Stenotic feces in round balls, cylinder (pipe-stem) or tape-like movements, are suggestive, but not conclusive. This type of feces may occur in spastic constipation, and, on the other hand, the feces may be normal in form with a stricture high up. Diarrhea at times alternates with constipation. The diarrheal movements may be extremely offensive and contain mucus, or even pus and blood; *this last is especially true in malignant stenosis*, where there are active ulcerations, or in intussusception. The diarrhea often relieves the patient. We must remember that chronic diarrhea is present in some cases when there is a *marked catarrh* above the stricture, and this will sometimes lead one astray. *Severe pain* of a colicky type occurs in all cases, and this may at times be excruciating; it may be localized near the seat of stricture, but in other cases be more diffuse, radiating even toward the thorax and producing a feeling of oppression and dyspnea. A symptom which occurs with the colic, which can be considered pathognomonic, is the visible *peristaltic movement of the intestines*[1], in which the loops can be seen to stiffen and relax alternately. The coils appear and disappear.

Vomiting may not occur at first, but is later more frequent, and if finally the obstruction becomes complete, may be marked and even feculent. Gurgling, rolling sounds, and meteorism are present.

Location of the Obstruction.—*This influences the character of the symptoms.* If the stenosis is situated above Vater's papilla, the symptoms are similar to those of stenosis of the pylorus. Marked dilatation of the stomach, nausea, and vomiting are prominent. R. T. Morris has demonstrated that in some cases of spider adhesions from the gall-bladder there may be severe hemorrhage and pain which may simulate gastric ulcer. There is usually a history of previous gall-bladder disease. If the obstruction lies below Vater's papilla, we again have gastric dilatation, but frequent bilious vomiting.[2]

The lower down the obstruction, the less pronounced the gastric symptoms, as a rule, and the more marked the constipation and colicky pains. There may even be an absence of gastric symptoms for a considerable period if the stenosis be in the lower ileum or in the colon. The symptoms, however, are always more severe in chronic intussusception or chronic types of strangulation than in ordinary cicatricial stenosis.

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[1] Peristaltic unrest (intestinal) is not always present, especially in the earlier stages of the disease; and at times, late in the condition, the bowel may become fatigued and paretic from overdistention. Often, however, the peristaltic movements will aid in locating the position of the obstruction.

[2] The stomach contents are *neutral* or *alkaline*, due to regurgitation of pancreatic juice and bile, and duodenal digestion takes place within the stomach. With stenosis above the papilla, gastric contents, as in benign pyloric stenosis, are acid. Riegel states another sign of duodenal stenosis is, that when the stomach has been emptied the night before, twelve hours later as much as 3 liters (quarts) can be frequently aspirated. The reaction and contents differ from that of gastrosuccorrhea.

X-Rays.—The administration of bismuth by mouth and inspection the following day with the fluoroscope or by photography may demonstrate the location of the stenosis. If it be apparently in the large intestine a few days later, it has been suggested that a high injection of bismuth and olive oil be given, and the examination be repeated as a check test. These procedures are not to be recommended in acute cases.

Inspection of the Abdomen.—When the stenosis is high up in the small intestine there is apt to be distention of the epigastric region, and when in the lower part of the small intestine or in the large intestine, then there is considerable abdominal distention.

The active peristaltic contraction of the intestines is marked, the coils (stiffened) rising and falling, and often performing winding or vermicular motions. These movements are associated with colicky pains and with gurgling and rolling noises.

Sausage-shaped ridges may appear with depressions in their vicinity, and in a few seconds the ridges disappear in one part and appear in another, the coils never remaining visible in one place for any length of time. They appear hard and stiff to the hand and then suddenly become elastic.

The contractions in the small intestine are usually smaller than in the large intestine.

Meteorism in the milder cases is not marked, as the gas can pass the obstruction. Later it may become quite marked, and, as in acute obstruction, may be local or general in character.

If the obstruction is in the lower colon or rectum, it will be most pronounced at first in the course of the colon, on the sides of the abdomen, and in the epigastrium.

If in the lower ileum, the distention is more pronounced in the umbilical and hypogastric regions, and the lumbar regions are relaxed (undistended).

Later there may be more general distention and the *barrel-shaped* abdomen. Local manifestations of meteorism are described under Acute Obstruction.

One of the important types is chronic intussusception, which may develop after an acute attack has subsided or may occur as such from its incipiency. It takes place most frequently in the ileocecal form. Some of the latter cases may continue for months or even years. The pain is paroxysmal, but may entirely intermit. There may be attacks of pain daily, or every few days or weeks. As the disease advances the intervals grow shorter. Vomiting is not marked. Often diarrrhea is present or constipation alternating with diarrhea. Blood may be passed and tenesmus is at times present. Palpation shows the presence of a tumor in about 50 per cent. of the cases, or a tumor can be felt in the rectum. Local meteorism may be present. Occasionally the invaginated part may slough off and perfect recovery ensue or, again, ultimate stenosis may follow. Death may occur through perforation.

Chronic Obstruction through Fecal Accumulation.—This is more common in females and usually in our older patients. The history is of habitual constipation. At times large amounts of fecal matter are voided by enema. Scybalæ are frequently present. There are digestive disturbances, flatulence, loss of appetite, eructation, fetid breath, headache, dizziness, and symptoms of autointoxication; there may be oppression in breathing from distention, an unhealthy appearance of the skin, and a foul tongue. There may be pains in the thighs, legs, and genitals due to pressure on the lumbar or sacral nerves. The patient may be very neurasthenic.

Distention and gurgling may occur. There are colicky pains, but usually they are not severe.

If untreated, the condition may become worse, the constipation increase, and all the symptoms of chronic or even of acute obstruction develop, as already described under Fecal Obstruction. The vomiting may even become feculent.

Palpation shows the presence of a tumor frequently in the colon, especially in the cecum, sigmoid, or other flexures. It may be hard and uneven, and will often "pit" on pressure. It is not painful on pressure, as a rule. Rectal examination generally shows the presence of scybalæ. I have treated a case in which apparently nearly the entire abdomen was occupied by the fecal tumor. Operation for "tumor" had been advised. As a result of 80 bowel movements in one week the tumor disappeared. The patient had had no bowel action for three weeks. Enemata, and especially recurrent rectal irrigation, and later, catharsis will reduce the size of the tumor. I have seen cases in which acute flexions or angulations of the sigmoid and colon have been factors in the production of this condition. They are well described by J. P. Tuttle.[1]

Rectal Stricture.—There are the symptoms of progressive constipation at times with alternating diarrhea, with mucus and pus in the stools, colicky pains, tympanites, tenesmus, and loss of appetite. There may be hemorrhoids and rectal prolapse. Digital examination, exploration with a rectal bougie or the proctoscope, will demonstrate the constriction.

With stricture the pressure on the examining finger remains constant and is not like sphincteric spasm, which soon relaxes. Many of the strictures are within the finger reach, within 4 to 6 centimeters up the bowel; if not within reach but suspected, a Wales rectal bougie or soft tube can be employed for the examination. The degree of stricturing can be determined by using tubes or bougies of varying sizes. It is preferable to pass the speculum up to the point of stricture, so as to examine its nature thoroughly. It is often advisable to remove a small section under cocain for microscopic examination. With malignant stricture there are cachexia, loss of weight, metastases, and the symptoms described under Carcinoma.

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[1] New York Medical Journal, etc., March 14, 1908. Gant has also demonstrated that prolapse of the sigmoid flexure is a cause, and relief has been secured by suspension of the sigmoid.

Complications.—Above the stenosis on account of the ulceration we may have circumscribed peritonitis and abscess or perforation with general peritonitis. The abscess may rupture into other viscera or perforate the abdominal wall. The chronic condition may suddenly become acute, and severe collapse occur with death, or thrombosis of some of the veins, or pyemic processes, or the patient may die of inanition.

Diagnosis.—The gradually increasing constipation; colic attacks with frequent stoppage of the bowels, alternating at times with diarrhea and the temporary relief of symptoms; visible peristaltic movements with tetanic rigidity of the intestines; at first local meteorism and, later, the tendency to the barrel-shaped abdomen; the presence of gastro-intestinal disturbances of varying degree and, frequently, loss of weight are suggestive of the obstruction. Rectal examination is always of importance.

If the constipation is of *long standing*, in an elderly person or invalid, and there is no cachexia, the tumor movable and doughy on pressure, scybalæ being passed, and at times accumulation felt in the rectum, fecal tumor is evident. These occur chiefly in the caput coli, sigmoid, colon flexures, or rectum.

With carcinoma there is marked cachexia[1] and the tumor is hard and solid, occurs mostly in the caput coli, sigmoid, and rectum, and more frequently in persons over forty-five. There is slight or moderate leukocytosis, and also anemia.

Tuberculous tumor of the cecum and peridiverticulitis (chronic) of the sigmoid must be held in consideration as causes.

With chronic intussusception the mass is usually of sausage shape, and shows the peculiarity that it sometimes feels hard and sometimes soft on palpation; there are mucus and blood in the stool.

External tumors can generally be appreciated. Vaginal examination should be made. If there have been attacks of peritonitis, bands and adhesions would be suspected.

A previous history of diarrhea, dysentery, syphilis, or tubercular difficulty would suggest ulcerative stenosis.

Stricture of the small intestine is most frequently due to adhesions or tubercular ulcer. In the large intestine stercoral, syphilitic, or dysenteric ulcers are to be considered, chiefly carcinoma or pelvic inflammation, perityphlitic adhesions, and, more rarely, tuberculosis of the cecum and chronic peridiverticulitis.

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[1] In the scirrhous type of carcinomatous stricture, cachexia may be scarcely noticeable for a considerable period. The progressive constipation and age of the patient are significant.

Course and Prognosis.—This depends on the etiology and severity of the obstruction. With non-malignant stricture of the bowel of moderate type, or in the fecal obstruction cases, not progressive, the patient by proper regulation of diet may live many years. With malignancy the prognosis is fatal unless relieved by early operation; chronic intussusception occasionally clears up, but the prognosis, as a rule, is bad. In progressive cases the symptoms rapidly become worse and life is shortened, with death from final acute obstruction. Unless radical operation is performed, complications such as peritonitis, pyemic processes, etc., hasten the final result.

Treatment.—If chronic obstruction suddenly or gradually develops into acute obstruction, the same indications for treatment exist as in the latter condition.

In the chronic cases the proper regulation of diet, omitting those things which will mechanically fill up the intestines and a careful regulation of the bowels, are most important. Substances that give a large residue of fecal matter, which are irritating and extremely constipating, should be excluded.

Matzoon, kumyss, buttermilk, bacillac, lactone-buttermilk, and kefir milk are excellent. Milk agrees well, as a rule, with most cases and is readily digested, while others it constipates and is undigested.

Raw eggs can be beaten up in milk, and soft-boiled eggs, broths, soups, and gruels administered. Sanatogen (flavored) and somatose are of value, administered in the broths or milk.

If the stenotic symptoms are progressive or fairly marked, liquid or soft diet alone should be given.

In milder cases scraped beef, tender meat (well divided), butter, a small amount of well-toasted bread, and moderate in quantity, rice, sago, and mashed potatoes in small amount are admissible. The patient should eat a small quantity frequently and should take sufficient food to preserve his nutrition. Very hot and cold drinks should be avoided. Irritating food, such as mustard, spices, pepper, vinegar, fruits in bulk, and green vegetables in large amounts, should be forbidden. Substances giving a large residue of fecal matter should also be cut off. Spinach I have found of service to aid bowel action. Fats, such as cream and butter, are useful.

Fresh fruit juices are valuable, and the administration of a glass of water on rising is of service.

Bowels.—The bowels must be moved every day. Injections of soapsuds enemata of medium size, not over 1 quart (liter), with the hips elevated; Kussmaul's method of oil injection, Oj (500 cc.) or more, being retained over night; or the addition of olive oil to the enema, mild cathartics, such as cascara, rhubarb, syrup of figs, phenolphthalein, and occasionally sulphate of magnesia or Apenta water, are of service. Under Chronic Constipation numerous remedies are described.

Enteroclysis (recurrent) is valuable in all cases.

Massage, vibratory massage, and electricity are of service only in *cases due to fecal accumulation.*

If diarrhea is present, unless the patient is weakened thereby, it should not be checked. In the latter event mild preparations, such as bismuth subnitrate, chalk mixture, or chalk and catechu, are preferable to opiates. If there are small diarrheal movements, evidently a diarrhea associated with constipation, then a dose of castor oil or a saline cathartic is indicated.

For Colic Attack and Peristaltic Movements.—Hot applications to the abdomen are indicated and a recurrent irrigation of normal saline solution at 115° to 120° F. is of value, to be given for ten to twenty minutes. The latter removes gas and clears the bowels. Enemata also can be administered.

Tincture of belladonna, ♏x (0.59), once or twice, is of use to allay spasm, or by suppository, extract of belladonna, gr. ¼ to ⅓ (0.016–0.024). Opium I avoid if possible, and then only ⅛ to ¼ gr. (0.008–0.016) of morphin or codein by hypodermic. It should be given only to allay severe pain.

For vomiting and distention lavage is indicated.

Cerium oxalate with bismuth and soda will check vomiting, also ♏j (0.059) doses of Fowler's solution of arsenic every hour for four doses.

Cocain, which has been suggested, is a dangerous and pernicious drug. I have seen complete collapse after its use in as small doses as $\frac{1}{10}$ gr. (0.006).

Lavage is of special value in temporarily allaying symptoms if the stenosis is in the small intestine.

If chronic fecal impaction, our first efforts should always be directed from below.

Hardened scybalæ should be removed from the rectum by the finger, oil injections, and, later, by soapsuds enemata and recurrent rectal irrigations. Olive oil can be given by mouth.

Later, cathartics, massage, electricity, and vibratory massage are of service.

Electric enteroclysis I have found useful in obstinate cases of fecal impaction.

Tincture of belladonna, ♏x (0.59) three or four times a day, and strychnin, gr. $\frac{1}{30}$ (0.002) t. i. d., are of value. Eserin sulph., gr. $\frac{1}{100}$ to $\frac{1}{60}$ (0.00065–0.00108), may be required.

Mild cases of rectal stricture, *providing they are not malignant,* can be benefited by dilatation for ten to fifteen minutes every two to three days with different sized bougies. Operation on the stricture may be necessay in some cases. Thiosinamin can first be tried, injected into the bowel or by hypodermic. Dose, gr. $\frac{1}{2}$ to $1\frac{1}{2}$ (0.033–0.1); or by hypodermic, gr. 1 (0.065), in 15 per cent. alcohol solution or 10 per cent. glycerinated solution.

Operation.—Most types of chronic intestinal obstruction grow worse, except those due to fecal impaction. In mild types of rectal stricture local dilatation may be palliative and keep the patient comfortable.

Malignant growths must be extirpated as soon as possible. Strictures must be treated according to their location—in the rectum, by division and dilatation or by resection. In other regions, enteroplasty, splitting the gut parallel to its axis, at the same time dividing the stricture and uniting the incision transversely, has been successful. Complete excision may be necessary, with or without employing Murphy's button to unite the ends. Anastomosis of the bowel above to that portion lying below the stricture may be required. In stricture of the colon in cases quite prostrated a simple colotomy above the point of stenosis is indicated.

Adhesions should be severed and tumors compressing the bowel removed. If the chronic obstruction be due to any of the causes which may also produce acute obstruction, appropriate surgical measures are indicated.

Early operation is preferable in most cases of chronic obstruction.

In acute obstruction engrafted on chronic stenosis, there should be immediate resort to surgery.

CHAPTER XXXII

NERVOUS DISEASES OF THE INTESTINES

UNDER this heading are included those conditions due to perversion of the innervation of the intestines independent of anatomic lesions of the intestinal wall or of distant organs.

In many cases the neurotic manifestations in the intestines are an independent manifestation of some general neurosis, such as of neurasthenia, hysteria, or hypochondriasis. Some few cases result purely from functional perversion of the intestinal nerves.

Intestinal neuroses may be divided into motor, sensory, and secretory. They often exist in combination. Psychic influences, such as fear, fright, worry, and anxiety, may be causes, as may reflexes from some diseased organs, such as the stomach or genito-urinary tract.

The nerve-centers influencing peristalsis, Meissner's and Auerbach's plexuses, have been described in the chapter on Physiology of Digestion, Part I, under Nervous Control of Peristalsis, page 41.

Secretion seems to be dependent to a great extent upon the ganglionic plexus. Moreau[1] ligated an intestinal coil and severed all the nerves passing to it. In a few hours it was filled with fluid, showing amylolytic qualities and containing albumin.

After ingestion of food into the stomach, secretion takes place in the lower part of the intestines before the arrival of the chyme. This was demonstrated by Quincke and Demant.

Vasomotor filaments exist in the intestines as stimulation of the splanchnic causes contraction, and its section causes dilatation of the intestinal blood-vessels. They are also concerned with absorption.

Sensory filaments exist in the intestines, since stimuli of greater intensity than normal, such as the ingestion of beans or cabbage, may give rise to sensations of pain or pressure. Kast and Meltzer[2] have demonstrated experimentally that the sensation of pain exists in the intestines, and that laparotomy, under cocain, causes anesthesia of the intestines through the cocain being carried by the blood.

MOTOR NEUROSES OF THE INTESTINES

Peristaltic Unrest (Tormina Intestinorum).—This condition consists in marked rotary or rolling movements of the intestines, so that they frequently become visible. It is usually seen in patients with hysteria or hypochondriasis. Occasionally it is an independent affection. It is almost exclusively seen in the small intestine.

[1] Centralbl. für die Med. Wissensch, 186, No. 14.

[2] Medical Record, Dec. 29, 1906.

Peristaltic restlessness, which accompanies complete or incomplete occlusion of the intestines, is not included herein. There is not the peculiar stiffening of the intestinal coils, as is present with stenosis, and other symptoms of that condition are absent. Occasionally the condition occurs in persons presenting no other nervous symptoms, as after the ingestion of highly spiced or indigestible foods, after the excessive use of tobacco, mental excitement, or too much brain work.

Clinically, there are rolling, gurgling, squelching noises in the abdomen of varying intensity. They can often be heard at some distance and are a source of mortification to the patient. Pain, as a rule, is not present. The movements of the intestines may occasionally become visible and palpable. Eructations sometimes occur when peristaltic unrest of the stomach is associated.

Attacks occur at irregular intervals and may take place during menstruation.

Diagnosis.—Stenosis of the bowel must be excluded. The nervous type of peristaltic restlessness of the intestines is readily recognized. Prognosis is favorable.

Treatment.—This should be directed toward the tone of the nervous system. Heat externally applied and the drinking of hot water during the attack are of value. Spicy and indigestible food should be excluded. Priessnitz compresses should be applied to the abdomen at night. The bromids, valerian, and asafetida are useful. Arsenic alone or combined with iron should be employed in anemia. If there is any disturbance of the bowels (diarrhea or constipation) it should be properly regulated. Rarely a small dose of opiate, alone or combined with belladonna, is required. If the attacks occur at night, chloral hydrate, gr. 15 (1.0), or veronal, gr. 7½ (0.5), sulphonal or trional, gr. 10 (0.6), may be necessary. Electricity and massage have been recommended. Change of climate is beneficial.

Nervous Diarrhea.—The exaggerated peristaltic movements occur not only in the small but also in the large intestine. They may be limited to the colon in some cases. There is an increased transudation of fluid due to nervous influences. The reader should refer to a description of this condition under Diarrhea (page 474). Spastic constipation, due to local enterospasm and also spasm of the sphincter, sometimes occurs in neuropathic, hypochondriac, or hysteric subjects. These conditions are described under Constipation (page 461).

Paralysis of the Intestines.—Paralysis resulting from a mechanical obstacle to the passage of the intestinal contents has been described. Primary paralysis of the intestines without any organic obstacle will itself cause symptoms of obstruction. The reader is further referred to dynamic ileus (page 651).

There are several forms of this condition: 1. An intestinal coil may become paralyzed after forced reposition of a hernia or after incarceration; it may be due to direct traumatism, to abdominal operation, or to inflammation or ulcerative processes of the intestines.

2. It may result from reflex irritation of the inhibitory nerves of the muscular coats of the intestines, especially where there is injury or inflammation which does not necessarily involve the bowel.

Toxemia may be a factor. Contusion of the testicles, abdominal abscess, anesthesia, uremia, etc., are causes.

3. Neuroses, melancholia, hypochondria, or affections of the nervous system, such as meningitis, brain tumors, tabes, myelitis, etc., are also causes. Atony of the intestines, leading to coprostasis, has been suggested as a cause of intestinal paralysis. It would seem that the symptoms are produced by occlusion. Intestinal atony is really a subparetic condition, and the nervous type is described under Chronic Constipation (page 460).

Meteorism in hysteria is probably due to sudden paresis of the muscular coat of the bowel. (See Meteorism, pages 451 and 452.)

Treatment.—Removal of fecal impaction by the fingers if present, enemata of soapsuds containing olive oil, ℥viij (250 cc.), and glycerin, ʒij (8.0), electric enteroclysis, enteroclysis, enemata, massage, and electricity are useful. With fecal impaction, mercury, ℥10 to 20 (300.0–600.0), given *through a stomach-tube,* is of value. It might otherwise enter the larynx. Various cathartics, such as castor oil, ℥j to ij (32.0–64.0), olive oil, ℥iv (128.0); physostigmin sulphate, gr. $\frac{1}{100}$ to $\frac{1}{60}$ (0.0006–.001), may be employed. Lavage, followed by the administration of the cathartic through the stomach-tube, is useful.

For further treatment, the methods pursued in dynamic ileus should be consulted (page 654).

Paralysis of the Sphincters.—It occurs as one of the symptoms of rectal affections. Tenesmus may lead to exhaustion. Ulceration and infiltration of the rectum at times involve the sphincters, interfering with their function or destroying it.

Improper methods of operations on the rectum may cause paralysis. Accumulation of feces may impair the tone of the muscles. Diseases of the brain and spinal cord may cause paralysis of the sphincters. *It may be a pure neurosis.*

Some patients are not able to keep the rectum tightly closed and a small amount of discharge continually escapes. In others involuntary movements occur after excitement, exertion, or during urination, there being only a partial paresis. With complete paralysis, flatus and feces escape involuntarily, even when resting.

With paralysis resulting from proctitis, hemorrhoids, stricture, etc., there is a continuous dripping of mucous secretion which irritates the skin.

Diagnosis.—The anus appears patulous and several fingers can be introduced into the rectum without resistance. To *diagnose purely* *nervous paralysis, anatomic lesions must be excluded by means of examination with a speculum.*

Prognosis.—This depends upon the cause—in the pure neuroses it is favorable.

Treatment.—Thorough evacuation of the bowels, preferably by enemata twice daily, is important. The addition of alum, ʒj (4.0) to Oj (500 cc.) of water by enema, is useful. If due to nervous conditions, electricity and massage, especially local vibrations, as suggested by J. P. Tuttle, are beneficial. Tonics, such as iron and arsenic, are useful. Strychnin, gr. $\frac{1}{60}$ to $\frac{1}{50}$ (0.001–0.0015), by hypodermic into the anal folds has been recommended by Rosenheim. General improvement of the nervous system and at times change of scene are indicated.

Occasionally difficulty in urination and straining may cause paresis of the sphincter. Catheterization will improve this condition.

In the cases in which the nervous condition is *not responsible,* but *some anatomic lesion,* appropriate treatment is indicated.

SENSORY NEUROSIS OF THE INTESTINES

I agree with Riegel that true colic is not a sensory neurosis. On page 453 I have called attention to the fact that the pain of colic is produced by tetanic contractions of the intestinal muscles, and that it is a secondary symptom.

Hyperesthesia of the Intestines.—Under normal conditions digestion is carried on without producing any sensation whatever. In cases of neurasthenia, hysteria, and hypochondriasis the patient may be conscious of abnormal sensations in the intestines after the ingestion of food. They may occasionally appear after violent emotion or shock. These sensations consist in a feeling of fulness, stabbing, burning, tearing, and as if the ingesta were moving about in the abdomen. Occasionally delusions may develop. In some there is local hyperesthesia, especially in the rectum. There is a feeling of tenesmus or fulness, as if some foreign material were impacted therein, though the rectum is normal and contains no fecal matter. In others pressure and weakness occur in this region, or there is burning, tickling, itching, stabbing, or a cutting feeling, at times combined with voluptuous sensations.

Anesthesia of the Rectum.—In these patients the desire for defecation is absent. In pronounced cases movement may occur without being felt. Such conditions are met with only in patients with spinal and brain trouble, or in the old and decrepit. Paralysis of the sphincters may occasionally accompany this condition.

Treatment.—This must be directed toward the improvement of the nervous condition. Change of climate and hydrotherapy are valuable. Highly spiced food, alcohol, and red meats should be forbidden.

Abnormal sensations in the rectum may be improved by cold rectal douches, sitz-baths, the cold prostatic cooler, such as I advocated in the treatment of hemorrhoids, and by rectal galvanization.

With rectal anesthesia cleansing enemata are useful. It may be necessary to wear a rectal obturator (Fig. 232) to prevent soiling.

Nervous Enteralgia (Neuralgia Mesenterica).—Aside from enteralgia due to irritating factors, it may result from a perverted state of the sensory intestinal nerves. This condition is not due to spasm of the intestinal muscles, like colic, but to a neuralgic affection of the bowels. It appears as a primary affection and is found in patients troubled with hysteria, neurasthenia, or spinal difficulty. It may occasionally be reflex, from abnormal conditions of the kidneys, bladder, uterus, ovaries, and liver. It may occur as a neuralgic condition even after the removal of some primary cause in the intestines. The first symptom is pain, which usually begins in the umbilical region, mild at first, but gradually increasing in intensity. It may radiate in various directions. Pressure over the abdomen and the passage of flatus usually relieve the pain.[1] The bowels may be nearly normal or constipated. Appetite and digestion may be good. Palpitation, dyspnea, strangury, etc., may occur.

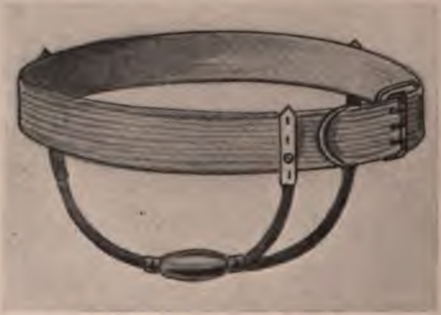

Fig. 232.—Rectal obturator.

Neuralgic attacks can occur in lead-poisoning without the true spasm. Hemmeter has reported 3 cases of gouty neuralgia of the intestines. Romberg holds that the abnormal crisis of tabes dorsalis is due to nervous enteralgia. Examination of the symptoms demonstrates that true colic is present. The Romberg symptom, Argyll-Robertson pupil, and absence of patellar reflexes are diagnostic.

Hypogastric Neuralgia (Romberg).—In some cases of tabes there is a purely local form of neuralgia, limited to the rectum. The attacks are characterized by violent tenesmus, paroxysms of pain, a feeling as if a red-hot iron were inserted in the rectum, and occasionally diarrhea. This condition is also frequently found in diabetes and in women having uterine trouble, piles, or who are neurasthenic.

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[1] The pain may be of a cutting or stabbing type, and may even produce shock or syncope.

The picture presented by nervous enteralgia, on the other hand, as Riegel remarks, may occupy an intermediate position between peritonitis and colic (pseudoperitonitis). Violent attacks of pain occur in the abdomen at short intervals. Associated with this is frequently pronounced collapse, with great abdominal tenderness on light pressure. Vomiting, which is usually present in peritonitis, is always absent in these cases. The skin of the abdomen is generally hyperalgesic. The functions of the intestines and stomach between attacks are undisturbed, and the patients feel perfectly well.

Treatment.—This should be directed toward improvement of the hysteria and neurasthenia. Change of climate, hydrotherapy, massage, electricity, and moral treatment are of service. Arsenic is valuable. The bowels should be kept regular and a simple diet advised.

With neuralgia hypogastrica, if there is local disturbance, this should be treated. Warm sitz-baths and hot enemata are useful. Occasionally an opium-and-belladonna suppository may be required. Tabes should receive treatment.

SECRETORY NEUROSES OF THE INTESTINES

Though secretion in the intestines immediately follows the entrance of food into the stomach, thus demonstrating the presence of secretory nerves in the intestines, we still have little knowledge of the subject.

Nervous diarrhea, which has been described on page 474 as a motor neurosis, is often accompanied by an increased flow of intestinal juice. Increased intestinal secretion is found in membranous enteritis, though I do not consider this disease *a pure neurosis* of secretion.

INTESTINAL NEURASTHENIA

Combinations of the intestinal neuroses frequently occur. Rosenheim designates such cases as intestinal neurasthenia. The appetite is good and the symptoms usually appear when intestinal digestion takes place—about two to three hours after meals.

There are pressure, tension, and griping in the abdomen. Occasionally there is nausea, and at times an evacuation of the bowels occurs accompanied with painful sensations in the abdomen and anus. Palpitation occurs at times; sometimes flashes of heat or cold. Generally the patient feels worse when resting in the recumbent position than when walking about. The symptoms usually disappear in a couple of hours to return later after a meal. Constipation usually is present.

The quality of the food *does not exert any influence on the symptoms.* Borborygmi and diarrhea occasionally are present, and the latter in the middle of the night or in the early morning. Indigestible foods are often well borne, while at other times small meals consisting of light food cause severe symptoms. Gastric neurasthenia is sometimes associated. This condition is found among the hysteric and neurasthenic.

Diagnosis.—Anatomic lesions causing these symptoms, intestinal dyspepsia, and enteroptosis must be excluded.

Treatment.—The general nervous condition must be toned up; iron, arsenic, strychnin, and the bromids are indicated, and ample feeding is required. Indigestible substances should be avoided, also red meats, to lessen the nervous irritability. The sour milks, such as bacillac, etc., are of value.

MUCOUS COLIC (MEMBRANOUS ENTERITIS)

Among the best-known synonyms for mucous colic are mucous colitis, membranous colitis, membranous or pseudomembranous enteritis, and tubular diarrhea. In all, there are about twenty-five names for this condition.

History.—Although no distinct accounts of this disease occur in the writings of the ancients, yet there may be detected some of its peculiar features in the description of certain pathologic conditions grouped under diarrhea, dysentery, etc. J. Mason Good, in 1825, was the first to classify the disease, calling it tubular diarrhea. Woodward descibed it in the "Medical and Surgical History of the War of the Rebellion." Siredy contributed a valuable paper in 1869. DaCosta, in 1871, described the nervous elements of the disease, stating that the condition is not a true inflammation. Leyden's work, in 1882, gave further stimulus to investigation, especially regarding the character of the dejecta. Nothnagel suggested the name "mucous colic," in order to show that a true enteritis need not exist. Mucous colic is, therefore, an entity, and may be defined as a "condition characterized by the excessive production of mucus in the colon, by attacks of painful spasms of varying degrees of severity and frequency, accompanied or followed by the expulsion of mucus in gelatinous masses, or in the form of tubular casts, or in tape-like pieces or strings, and, furthermore, characterized by anomalies of the gastro-intestinal functions and by various nervous symptoms."

Age and Sex.—It is a comparatively rare affection, occurring most frequently in women from twenty to forty, frequently in middle life. A few cases occur late in life and rarely in children. Boas reports one in early infancy. About 75 to 85 per cent. occur in women.

Etiology.—Space will allow me to mention only a few of the chief investigators. Among the various theories regarding the etiology, we may mention the following:

1. Neurasthenia is the prime factor—mucous colic is a secretory neurosis. Among the advocates of this view are notably DaCosta, Siredy, and W. Mendelson, of New York City.

2. The anatomic origin. Ewald lays stress on ptosis of the colon; Boas, on atony; Glénard, on splanchnoptosis.

3. Partly nervous and partly anatomic origin. Mathieu considers

it a hypersecretion of mucus in patients of a neuro-arthritic type, who suffer from enteroptosis, intestinal sand being present. Hemmeter believes that often there is some connection with arthritis. Von Noorden lays stress on long-continued constipation in nervous subjects. Einhorn places it among the neuroses, but finds that it is associated in many cases with Glénard's disease (with gastroptosis and enteroptosis), and that achylia gastrica is present in many patients.

4. Tumors, adhesions, enlarged prostate, and various other factors are given. J. P. Tuttle believes mucous colic due to organic causes. Roger traces the cause to the liver, believing there is an anticoagulant in healthy bile, and when its production is interfered with by visceral ptosis, abnormal accumulation of mucus begins.

Nepper[1] also imputes the condition to disturbance of the biliary functions.

Pathology.—Necropsies are rare unless death results from some intercurrent disease. Autopsies in the cases of O. Rothmann, Osler (Edwards), and Weigert demonstrate that no inflammatory condition existed in the colon. There was simply hypersecretion of mucus. The consensus of opinion is that no inflammation exists. On the other hand, M. Rothmann reports 1 case and Hemmeter 2 cases in which, in addition, some catarrhal inflammation was present. Nothnagel explains this unquestionably by the fact that there are two classes of cases, one in which there is the pure "mucous colic," with hypersecretion of mucus; and the second class, in which the mucous colic is engrafted on a catarrhal colitis. I have noted, in my own experience, that the catarrhal colitis may be of such a mild type that attention may readily be diverted from it on account of the predominance of the symptoms of the mucous colic.

The mucus may be passed in the form of long, thin bands, ribbon-like or in the shape of a tapeworm; they may be tubular or form a cast of the intestines; in some cases these are of considerable length, several feet; the mucus may be in jelly-like masses or even in shreds, occasionally streaked with blood. This discharge should be carefully differentiated from fascia, tendons, the membranes of oranges, etc. After first treating with sublimated alcohol and then staining with Ehrlich's triacid solution, *a green color occurs with mucus*, of which this discharge consists; with fibrin it turns red. The color of the membranes in mucous colic is ordinarily grayish, though they may be translucent or even transparent. Microscopically, the membrane consists of a structureless matrix, with columnar epithelium scattered therein; its chief constituent is mucus.

Symptoms.—These patients are markedly neurasthenic and morbidly self-conscious; in appearance they are usually emaciated, with a history generally of considerable loss of weight. There has been obstinate constipation of long duration, with an occasional intermittent diarrhea. Palpitation, dizziness, disturbances of the genito-urinary system, hysteric symptoms, anemia, headache, and gastric disturbances of various types are present.

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[1] Mucomembranous Colitis, its Causes and Mechanism, New York Medical Journal, May 23, 1908.

On palpation of the abdomen sensitive points will often be detected. Patients give a history of a sudden attack of acute abdominal pain like severe colic, and the abdomen may become swollen and tense. At this time the nervous symptoms become extremely aggravated. Finally, the passage of the mucous masses described occurs spontaneously, with great straining or with artificial aid. These attacks occur with varying frequency and severity. Between the attacks the nervous conditions of the patient may be slightly improved. This is the type of uncomplicated (pure) mucous colic.

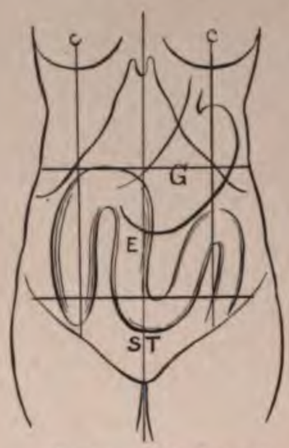

Fig. 233.—Mucous colic: Presence of gastroptosis (G) even of a mild degree, as demonstrable by gastrodiaphany shows enteroptosis (E) is present also; enteroptosis with sacculation and narrowing at ST; passive congestion and mucus accumulation occurs in enlarged (sacculated) portion of colon; mucous colic attacks occur as result.

Nothnagel describes a second type of enteritis membranacea which is engrafted on a colitis. He notes two classes of cases:

(*a*) That in which the severe *cramp-like attacks are absent;* the patient passes mucus continuously, with occasional tube-casts—a cystic colitis (Abercrombie's case), as shown at autopsy. This is not a true mucous colic.

(*b*) A class in which mucous colic is engrafted on a chronic catarrhal colitis—the latter due in this case to adhesions from recurrent appendicitis. There were small amounts of mucus passed at frequent intervals with occasional attacks of mucous colic. Operation relieved both conditions at first, but the mucous colic later returned.

I have had such a patient under treatment; the appendix had been removed and adhesions broken up. The patient improved for a time, but later relapsed. I found enteroptosis associated with gastroptosis. I applied Rose's belt and instituted treatment. Improvement immediately followed, with ultimate cure.

Researches.—For some years the author has carried on investigations in gastroptosis and enteroptosis at the Manhattan State Hospital, as well as other institutions, and also into the etiology of mucous colic,[1] and he is thoroughly convinced that enteroptosis is a factor in mucous colic.

In Fig. 233 is illustrated ptosis of the colon, narrowed at one point and sacculated above this. Gastroptosis is associated with it.

[1] American Medicine, vol. ix, No. 9, pp. 349–354, March 4, 1905.

This misplacement of the colon undoubtedly favors circulatory and, hence, secretory changes in the sacculated portion of the colon. Fecal accumulation is also favored, a further cause of irritation. Absorption, with resulting auto-intoxication and nervous disturbances following the same, can thus readily result. Naturally, a patient of nervous temperament, and there are undoubtedly many such, may be more markedly affected, but I do not believe that neurasthenia *per se* will cause that peculiar entity known as mucous colic any more than it will cause gonorrhea. There must be other factors.

We know that mucous colic occurs in Glénard's disease, that Ewald believes that ptosis, and Boas that atony, of the colon are important factors, and Einhorn finds a large percentage of patients with mucous colic that have enteroptosis associated with gastroptosis and achylia gastrica.

I believe that mucous colic has as its chief etiologic factor ptosis of the colon with associated gastroptosis.

I have had under observation a patient with typic attacks of mucous colic which began one month after confinement. She had enteroptosis and gastroptosis (Landau's disease) due to insufficient support of the abdomen after the birth of her child. She was not neurasthenic, and was only nervous at the time of her attack.

A specimen of the mucus passed by this patient was about 12 inches long, flat, and tape-like, and gave the typic reaction to the mucin test; no fibrin was present. To my knowledge, this is the first case of mucous colic that has been *reported without the usual accompaniment of neurasthenia*,[1] and it substantiates my views. In addition, in 9 cases of mucous colic which I have carefully examined since I began these special investigations, I have found in every case varying degrees of gastroptosis with its associated enteroptosis.

In 4 cases there was hyperacidity; 2 cases, anacidity; 3 cases, achylia gastrica. Since the publication of the above article the author has investigated many more cases, all of which substantiated this view.

In the newborn and in young children, in whom several cases of mucous colic have been reported, neurasthenia surely cannot be claimed as a cause of the condition. As before stated, *visceral ptosis* may be present and the patient be in perfect health; but some contributory factor, local irritation, anemia, or intercurrent disease may destroy the equilibrium, and gastro-intestinal disturbances, constipation, etc., may result, and finally, mucous colic. On the other hand, gastro-intestinal ptosis may be brought about by loss of weight or other factors mentioned under Etiology of Gastroptosis, and mucous colic finally result. Enteroptosis is not invariably productive of mucous colic, any more than is typhoid always complicated by hemorrhage or perforation.

Enteroptosis with associated gastroptosis with gastro-intestinal disturbances I consider factors in the production of mucous colic, and the neurasthenia the result of auto-intoxication. In effect, it may be considered as one of the manifestations of Glénard's disease. Other contributory factors, such as rectal irritation, associated colitis, etc., will be referred to under Treatment. In a paper entitled "A Consideration of the Etiology of Mucous Colitis," by John A. Lichty,[1] there are reported 21 cases of mucous colitis; ptosis of the viscera was demonstrated in 16 cases; the other patients were seen before the author's attention had been directed to splanchnoptosis. He states that it is a well-known fact that not infrequently during the examination of a patient a condition of ptosis is found without any symptoms referring to it. In such cases there has been established what may be called a condition of perfect compensation, and physiologic function has not been disturbed. When, however, this compensation is lost or disturbed, the symptom-complex of mucous colitis appears. He notes a lithemic condition in several patients—notably one having had several attacks of acute articular rheumatism. The gastric secretion was studied in 8 patients—in 4 it was hyperacid; in 2, normal; in 1, hypo-acid, and in 1 achylic.

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[1] Medical News, Aug. 6, 1904; American Medicine, March 4, 1905.

Prognosis.—These cases require tact and patience, and most of them are of long duration. With perseverance, I believe them to be curable.

Treatment.—This may be summarized as follows: During the attack, rest in bed; the application of heat to the abdomen by flaxseed poultices, turpentine stupes, or hot pepper poultices—ʒj (4.0) red pepper to Oj (500 cc.) of boiling water—a flannel being wrung out therein, covered with oiled silk, and applied to the abdomen. Dry heat, by means of a hot-water bag, salt-bag, or light tinplate (pie plate), heated in the oven and covered with flannel, may be employed. Spice poultices are of service. Moist heat, however, seems best.

The greatest relief to the cramps and bearing-down pains is afforded by enteroclysis by recurrent irrigation with normal saline solution at 110° to 120° F., oil of peppermint, ♏v to xv (0.296–0.888) to the quart (liter), may be added. Several gallons should be employed once or twice in twenty-four hours, and no fluid should be left in the bowel after irrigation, lest further cramps ensue. High enemata of warm olive oil—1 pint to 1 quart (500 cc. to 1 liter)—are also of service, as they aid in relieving spasm just as does the internal administration of olive oil in spasm or stenosis of the pylorus. Hot saline rectal injections containing ℥ij to iv (60.0–125.0) of milk of asafetida may be employed.

Diet.—Fluid diet, milk, broths, soups, etc., should be enjoined during an acute attack.

Medication.—Tincture of belladonna in doses of ♏x (0.592 cc.) every three or four hours, and pushing even to physiologic symptoms, has given me the best results in the treatment of spasm. Occasionally it may be necessary to employ codein in .016 to 0.03 gm. ($\frac{1}{4}$–$\frac{1}{2}$ gr.) doses, or even morphin, 0.008 to 0.016 gm. ($\frac{1}{8}$–$\frac{1}{4}$ gr.), in conditions of extreme pain. If the acute attack is rather prolonged, the internal administration of valerianates or of asafetida, and the addition of milk of asafetida to the enema may prove to be of value.

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[1] American Medicine, Aug. 6, 1902.

Between attacks I apply proper abdominal support. For this purpose a silk elastic abdominal supporter, the Van Valzah-Hayes support, Gallant's or La Grecque corset, or Rose's adhesive plaster belt can be used. Rose's belt has the advantage of simplicity and it cannot slip or become displaced.

My great object is to "put on fat" in all cases, and as ptosis of the colon and of the stomach are great factors in the disease, the increase of intra-abdominal tension should be secured by this means. The belt is an aid in the relief of the functional disorders of the stomach incident to the gastroptosis. If the patient objects to the plaster, then the silk abdominal supporter may be employed. In exceptional cases it may be necessary to resort to the rest cure, associated with hydrotherapy and electrotherapeutics. Under such conditions we may increase the weight by following out Russell's method, such as he first instituted at the Post-Graduate Hospital in the treatment of tuberculosis. It was advocated by me in cases of gastroptosis. Regarding the constipation, the Küssmaul-Fleiner method of injecting into the rectum nightly or every other night warm olive oil, to be retained all night, is of great value. At the beginning one may employ a few ounces, increasing it to 1 pint (500 cc.) or even 1 quart (liter). The patient should be taught regular habits in attempting bowel movement. A glass of hot or cold water administered an hour before breakfast is valuable as an adjunct. Fluidextract of cascara sagrada or the compound cascara tablets have been found serviceable, and in some cases sodium phosphate administered in the morning is of value. Other remedies, as suggested under Chronic Constipation, can be employed. A thorough bowel action should be secured daily. Massage of the bowel may be employed, massage with a cannon-ball, or vibratory massage. Sensitive areas must be avoided. These methods can be used while Rose's belt is *in situ*.

Enteroclysis several times a week with normal saline solution is useful, since it promotes intestinal peristalsis, prevents the accumulation of mucus, and lessens the chances of spasmodic attacks. With obstinate constipation electric saline enteroclysis is of value. In addition I sometimes employ baths, abdominal compresses, and electricity.

The carbonic acid bath (Nauheim) is of value in improving the circulation. For the nervous conditions the same bath (Triton salts) has proved of service. Achilles Rose has devised a simple method for administering the dry gas bath without the patient being obliged to disrobe (Fig. 234).

The tank is filled with gas and the patient sits therein. The height to which the gas rises is estimated by means of a burning candle, which goes out when the gas reaches that point.

I have secured at least one brilliant result in the treatment of mucous colic by inflation of the colon with CO_2 gas, as advocated by Dr. Rose. The method seems in some cases to improve the local circulatory conditions in the colon, just as it affects the peripheral circulation when the bath is given. I believe it worthy of trial as an adjunct to the other treatment.

It merely requires a bottle with a large mouth. A glass tube passes through the cork. To this tube is attached a piece of rubber tubing with a rectal tip. From ʒss to j (2.0–4.0) each of bicarbonate of soda and tartaric acid are placed in the bottle, which is then filled two-thirds with water. The CO_2 gas is thus generated, and the bottle being elevated slightly above the rectum the gas is allowed to flow in until slight distention is observed. This procedure can be carried out every other day. Rose's CO_2 bottle is illustrated in this volume.

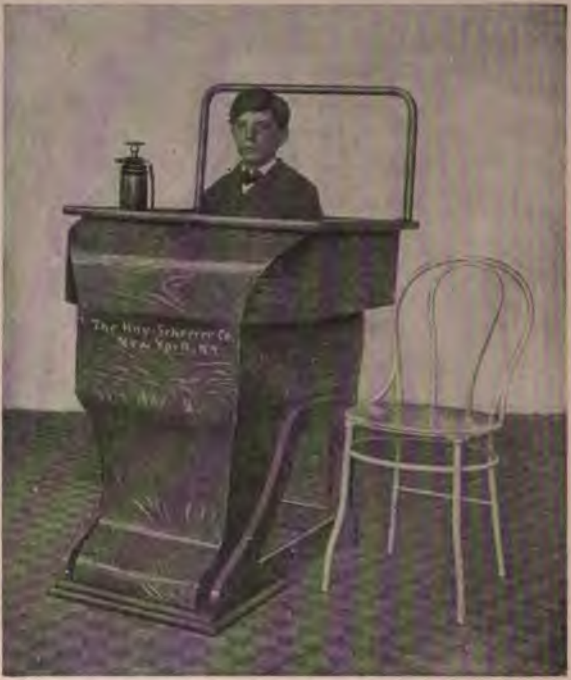

Fig. 234.—Rose's dry carbonic acid gas bath.

I have referred to certain mixed cases in which there was a catarrhal colitis with a mucous colic later engrafted upon it. Among such we can classify those that may apparently be caused by excessive bicycling or horseback riding, enlarged prostate, uterine fibroids, adhesions from appendicitis, etc. In some of these conditions a local congestion of the rectum or sigmoid can be detected, and careful investigation will demonstrate that the attack first starts as a simple proctitis or colitis. Constipation has been previously present. Subsequent auto-intoxication, nervous symptoms, and mucous colic result. I believe that careful examination will reveal that these patients have had an existing ptosis of stomach and colon, quiescent, with no resulting symptoms, but as a result of irritation causing favorable conditions, mucous colic develops. The correction of such sources of irritation is undoubtedly rational, and will thus readily explain the improvement which at all times occurs after operative procedure, as in the chronic appendicitis case with colitis and mucous colic previously described. The existence of the "mixed cases" will undoubtedly "clear up" the hitherto apparently diverse opinions as regards the etiology of this disease.

In such cases, with a coexisting catarrhal colitis, irrigation with nitrate of silver, 1.3 to 2 gm. (20–30 gr.) to 2 quarts of water, and followed by saline solution, or with resorcin, .65 to 1.3 gm. (10-20 gr.) in 2 quarts, or with listerin, borolyptol, glycothymolin, 4 to 8 gm. (1–2 dr.) to 2 quarts, or with gomenol, 4 gm. (1 dr.) in the same quantity of water, may prove to be of service. I have often found enteroclysis with demulcents, such as weak flaxseed tea, or 185 to 250 cc. (6 to 8 oz.) of a saturated solution of gum-arabic added to 2 quarts (liters) of warm water, of value.

In pure mucous colic I employ only normal saline solution or the demulcents for removal of the mucus, since the condition is due to hypersecretion and not to inflammation. I should avoid silver irrigations in such cases, since I have already referred to the fact that irrigations of silver, tannin, alum, etc., can produce an artificial hypersecretion. Small doses of olive oil or of castor oil, in capsules, seem of value for constipation, and improve the tone of the mucous membrane of the intestines, providing they do not increase the patient's dyspeptic symptoms.

Extract of nux vomica, gr $\frac{1}{4}$ (0.016), or strychnin, gr. $\frac{1}{60}$ (0.00108) t. i. d., is of service in increasing the tone of the gastro-intestinal tract and the general muscular system. Resorcin, 5 gr. (0.3), or sodium benzoate, 5 to 10 gr. (0.3–0.6), or bismuth salicylate, 5 to 10 gr. (0.3–0.6), should be given if there is much gastro-intestinal fermentation. The use of the following, suggested by W. H. Thomson in the mixed cases for the treatment of catarrh, gives good results:

℞. Silver nitrate.............................. 0.32 gm. (gr. v)
Resin of turpentine........................ 8.0 gm. (ʒij)
Potash solution.......................... 4.0 gm. (ʒj)
Pulverized licorice, q. s. to make pills soft.—M.
Divide into 60 pills.
Sig.—Three pills t. i. d.

Copper sulphate, 0.016 ($\frac{1}{4}$ gr.) t. i. d., may be substituted later; Fowler's solution of arsenic in .06 cc. (♏j) dose t. i. d. has also been found useful in these mixed cases.

General Treatment.—Exercise and outdoor life, as golf, etc., to strengthen the abdominal muscles, are important. During winter weather fencing is useful. The general nervous system must be toned up and anemia should be corrected. Iron tropon is easy to assimilate. An excellent combination is a fresh Blaud's pill (iron), .32 gm. (5 gr.), made soft with honey; in each pill is incorporated ♏ij (0.118) Fowler's solution of arsenic, and extract of nux vomica 0.008 gm. ($\frac{1}{8}$ gr.), or 0.00108 gm. ($\frac{1}{60}$ gr.) of strychnin. The glycerophosphates or phosphorus compounds are of value for the nervous conditions. Hydrotherapy, massage, and electrotherapy may be used.

Diet.—As before noted, fluid diet, milk, kumyss, bacillac, lactone-buttermilk, broths, gruels, etc., with the addition of somatose or liquid peptonoids, should be used during the attacks. Between attacks, Von Noorden advocates a very coarse diet (bread containing plenty of chaff, vegetables rich in cellulose, fruits with skins, etc.), to form ballast for the bowel. He claims excellent results. It is my *custom to determine the condition of the stomach.* Like Einhorn, I have found cases of achylia gastrica in mucous colic, but more cases of hyperchlorhydria and a few of hypochlorhydria. These special conditions should be treated in each individual case and appropriate diet instituted. Stomachics and dilute hydrochloric acid should be given when there is deficiency of HCl, and alkalis if there is hyperacidity (see Hyperchlorhydria, Achylia Gastrica, etc.). We should, however, give our patient abundant nutrition. Cod-liver oil and fats, such as Russell's emulsion, cream, etc., are of value when they can be assimilated. The addition of healthy fat, with increase in weight, means the cure of our patient.

Surgery.—Some writers, notably Hale White, have recommended a right inguinal colotomy to give rest to the colon in certain intractable cases. This would not relieve the ptosis, however. In severe cases one might resort to shortening the suspensory ligaments of the stomach and colon. Gastropexy and colopexy might be performed, but to my mind it is always objectionable to suture a viscus to the abdominal wall. If there is hepatoptosis, Elliot's operation for support of the liver might be instituted at the same time. These procedures will aid in the support of the floating kidney if such be present. Nephropexy, I believe, is rarely indicated when it is a part of general ptosis, unless there be some evidence of nephritis or interference with its functions. Some recommend a "revision" (tightening) of the abdominal muscles (recti) by means of suturing, so as to relieve the muscular relaxation. This last procedure, as advocated by R. T. Morris, I consider preferable. I believe that resort to surgery is rarely required except in the most obstinate cases, and only after at least two years' continuous medical treatment, with failure to secure cure or comparative comfort for the patient.

CHAPTER XXXIII

INTESTINAL PARASITES

MOST of the animal parasites that occur in mankind inhabit the intestinal canal. There are about fifty varieties, but all do not produce morbid conditions. Some cause a pathologic state locally in the intestines or by their toxins in the blood. There are no absolutely characteristic symptoms produced by these parasites, but they are detected by discovering either them or, in the case of worms, their ova in the stools.

Gastro-intestinal disturbances, with or without anemia and with nervous symptoms, may result from their presence. There are two chief groups—the protozoa and the vermes.

PROTOZOA

AMEBÆ

In addition to the dysenteric amebæ which have been described, amebæ differing slightly in certain characteristics from the dysenteric variety have been reported. They are said to give rise to no symptoms or at times to slight diarrhea. Musgrave is skeptical as regards the existence of non-pathogenic amebæ.

SPOROZOA

Coccidia are occasionally found in the stools. These are egg shaped, provided with a thin shell, are about 0.02 mm. long, containing in the interior a large number of nuclei, usually arranged in groups. They do not seem to have a pathologic bearing.

INTERNAL PSOROSPERMIASIS

Psorosperms have been found in the liver, spleen, kidneys, and ileum, producing a disease similar to that in rabbits. One patient, notably referred to by Osler,[1] was thought to be suffering from typhoid fever. The patient had diarrhea and enlarged liver and spleen. Masses resembling tubercles in the liver, spleen, and ileum were found to contain coccidia.

INFUSORIA

Cercomonas intestinalis is pear shaped, has a distinct nucleus, and eight flagellæ. The head tapers obliquely and has a depression

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[1] Practice of Medicine, 1906.

(Fig. 235). It is believed that this organism is liable to prolong existing catarrhal affection of the intestines.

Trichomonas intestinalis is distinguished from the former by its greater size and the row of fine cilia on its periphery (Fig. 236). In fresh dejecta it shows active movements. Zunker[1] reports it in mushy dejecta of yellowish-brown color and putrid odor.

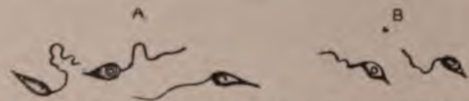

Fig. 235.—Cercomonas intestinalis: A, Larger, B, smaller, variety (Davaine).

Balantidium (Paramœcium) Coli.—The body is oval shaped, measuring from 0.07 to 0.1 mm. in length, by 0.05 to 0.07 mm. in breadth (Fig. 237). The anterior end is slightly truncated, with a short peristome, generally funnel shaped, and opens externally near the anterior pole. When feeding it opens out and broadens, so one can see it is a mouth which leads to a gullet and not a simple furrow. The left border has long cilia, while the rest of the mouth is destitute of them. The surface of the cortical layer is surrounded by a cuticle covered with cilia. The interior structure consists of granular substance. It contains a nucleus and contractile vacuoles.

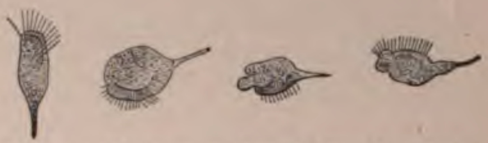

Fig. 236.—Trichomonas intestinalis (after Zunker).

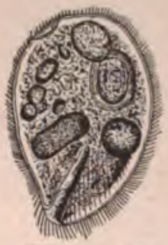

Fig. 237.—Balantidium coli (Malmsten).

Fat and starch granules and, occasionally, red and white corpuscles may be found within the granular substance. The posterior end is rounded and contains the anus. Particles may be observed to pass from it. The parasite can change its shape and possesses both forward and rotary motion. Reproduction occurs by division, budding, and conjugation.

The balantidium is a parasite of the colon and cecum of *the hog*. Human infection probably occurs most frequently through the infusorium entering its host in the *encapsulated state*. When hog feces are dried and broken up, the encysted forms are scattered about and come in contact with the food or drinking-water. The parasite has been found in the city of London in the drinking-water. The disease frequently occurs after the preparation of sausage or the ingestion of uncooked sausage-meat. Malmsten first described the disease in 1857.

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[1] Deutsche Zeitschr. f. Praktish. Medicin, 1878, No. 1.

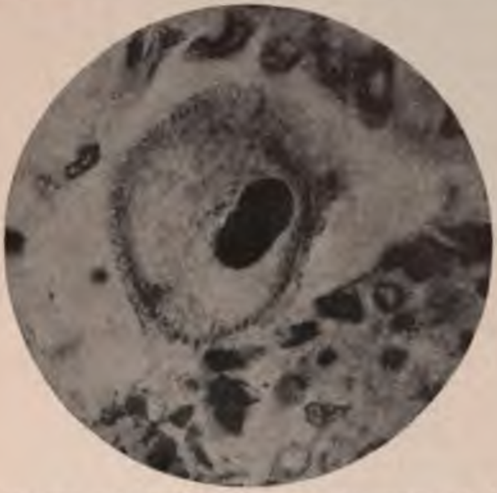

Fig. 238.—Balantidium coli: Parasite more highly magnified, showing flagella (Strong).

Musgrave has written on the subject, and for the most complete description the reader should refer to R. P. Strong's[1] article, "The Clinical and Pathologic Significance of Balantidium Coli." He tabulates 117 cases.

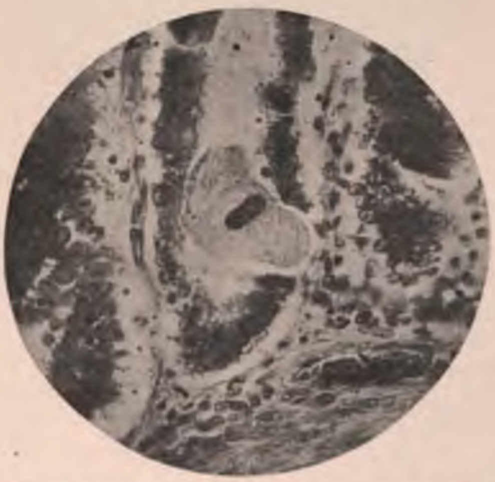

Fig. 239.—Balantidium coli: A parasite passing through the walls of a gland of Lieberkühn, rupturing the basement membrane. The parasite shows the striation referred to in the text (Strong).

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[1] Bureau of Government Laboratories, Manila, No. 26, Dec., 1904.

The balantidia frequently exist alone, though sometimes other parasites, such as Bothriocephalus latus, Ascaris lumbricoides, Trichocephalus dispar, etc., may be associated.

Harlow Brooks[1] found balantidia the cause of an epidemic of diarrhea among the orang-outangs in the New York Zoölogical Park. The lesions found in human beings seem to be an ulcerative colitis (Figs. 238–240), with the infection in the large intestine. In some cases there were swelling of the lymphatics of the mesentery and mesocolic glands and chronic adhesive peritonitis. Pulmonary, cardiac, renal, and cerebral complications may occur.

Symptoms.—The presence of Balantidia coli in the stools is usually associated with diarrhea; the feces are liquid, often contain mucus, sometimes undigested food, and frequently blood. Diarrhea is persistent until treatment is directed against the parasite. Colic is frequent, nausea and vomiting may occur. The abdomen may be swollen. It is often painful on pressure along the colon, and on palpation the latter may feel thickened. Tenesmus is common.

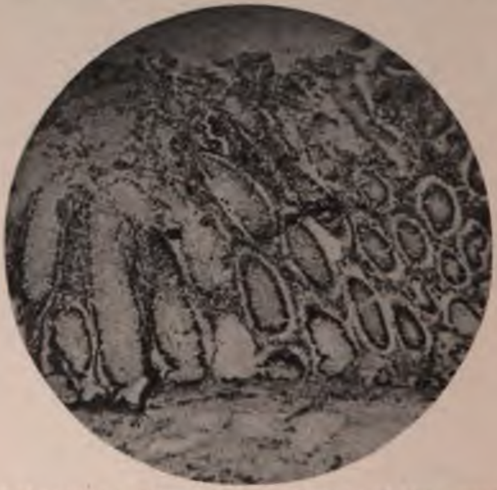

Fig. 240.—Balantidium coli. The early lesions of the mucosa, consisting of desquamation of epithelial cells of the glands, round-celled infiltration, etc. About a dozen parasites may be counted in this field (Strong).

In chronic cases there are weakness, exhaustion, and emaciation; more or less anemia and edema of the feet and ankles.

In the fresh stools the balantidia move about rapidly, but die in from one-half hour to two hours after the dejecta have been passed.

Only 2 cases are reported in children; 25 per cent. of cases give the history of association with or caring for pigs or having eaten or prepared fresh sausage.

Eosinophilia is present locally in the intestines and also in the blood. The temperature may be subnormal or at times of considerable height.

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[1] New York University Bulletin of Medical Sciences, Jan., 1902.

The disease has been found in Germany, Sweden, Russia, France, the United States, and the Philippines. Balantidia probably occur more frequently than we suppose.

The mortality was 30 per cent., but depended somewhat on the presence of other diseases which were associated in many of the fatal cases reported. In 33 per cent. there were cures, and improvement in the other cases.

Treatment.—The following solutions for topical irrigations or enemata have been suggested:

Ems' salt, by water enemata, gr. 15 (1.0) are added to 1500 cc. (1½ quarts) of water, followed by quinin enemata, 1 quart (liter) of 1:750 or 1:500 strength.

Others suggest calomel, gr. 2 (0.1) t. i. d. for two days only, or naphthalin, gr. 5 (0.3) t. i. d., daily by mouth, with tannic acid, ʒj (4.0) to Oj (500 cc.) of water, by injection, every day.

Acetic acid enemata—acetic acid, gr. 50 (3.3) to 2 quarts (liters) of water at 37° C.—once or twice a day, with tannic acid, gr. 5 (0.3) t. i. d., by mouth, proved useful in some cases.

Another combination is salicylic acid, gr. 15 (1.0), and sodium sulphate, ℥ss (16.0), morning and night, by mouth, with enemata twice a day of salicylic acid (1:1000), or salol, by mouth, gr. 5 (0.3), four or five times a day, with salicylic acid (1:1000), or boracic acid enemata ʒj (4.0) to the quart (liter) of water, are suggested.

Sodium bicarbonate (2 per cent.) enemata, followed by a salicylic acid (1:2000 to 1:1000) enema, with salol, gr. 5 (0.3), and tannalbin, gr. 5 to 10 (0.3–0.6), by mouth, each several times a day, have given good results.

Thymol (1:2500), acetozone (1:1000), quinin bisulphate (1:500), administered by enema or irrigation, as in dysentery, with the use of salol and tannin preparations by mouth, are valuable.

One might also administer small quantities of acetozone (1:1000) by mouth.

VERMES

Cestodes (Tapeworms); Hydatid Disease.—The adult parasites live in the small intestine of man; the larval forms, in the muscles and other organs.

The most important varieties of tapeworms found in human beings are the Tænia solium, Tænia mediocanellata, and the Bothriocephalus latus.

The symptoms produced by these varieties of tapeworms are about identical, except in the case of the bothriocephalus, which at times gives special symptoms.

The parasites are found at all ages, are not uncommon in children, and occasionally are found in sucklings. *They may cause no disturb-*

ance, and one may only learn of their presence by noting segments of the tenia in the dejecta.

In other cases there may be general as well as intestinal disturbances.

There may be pressure in the pit of the stomach, abdominal pains, ravenous appetite (bulimia), nausea, at times loss of appetite, occasionally vomiting. Diarrhea is sometimes present or there may be constipation.

In women and nervous patients we may see mental depression and even hypochondria. There may be dizziness, headache, fainting spells, chorea, convulsions, and even epileptic attacks. Some patients may emaciate, feel weak, and suffer from palpitation.

The bothriocephalus may cause a severe and even fatal anemia (pernicious), with poikilocytosis and nucleated red blood-corpuscles; and with it there may be edema of the feet and eyelids and hemorrhages from the mucous membranes.

The metabolic products of this worm probably have a hemolytic action.

The diagnosis can only be made by discovery of the segments of the parasites or their eggs in the stools.

General Description of the Tapeworm.—It has a scolex or head, which may live for years even when detached from the rest of the body, an oblong neck, and detachable segments (proglottides). These last vary in size and shape and possess the power of motion. The worm is flat and devoid of mouth or intestines. It grows by alternate generation through germination of a pear-shaped primary host (head), and remains united to the latter for a time as a colony of band-like shape. Each segment forms a sexually active individual. The proglottides gradually increase in size as they become more distant from the head, and then diminish again toward the extremity. The tapeworm is an hermaphrodite. On its head are four sucking disks, by which it attaches itself to the mucosa of the intestines. By means of pores it derives its nourishment from the chyme.

The older proglottides contain many fructified eggs. These are emptied at intervals into the intestinal canal and appear in the dejecta. The ovum contains an embryo, which requires for its development an intermediary host. After reaching the stomach the envelope is dissolved by the gastric juice. The embryo is set free and finds its way by the lymphatics or blood-vessels to some place (usually the muscles) where it settles. It here surrounds itself with a sac, which may later be surrounded by a calcareous deposit. In this condition it is called a cysticercus or measle. When the measle reaches the stomach of a new host it opens, and its scolex enters into the small intestine, where it develops into a full-grown tapeworm.

Tænia Solium.—Armed tapeworm or pork tapeworm. This is not common to North America, but more frequent in Europe and Asia. When mature it is from 6 to 12 feet (2–4 meters) or more long. The head is smaller than the head of a pin, spheric, and provided with four sucking disks, in the middle of which is the rostellum and a double row of hooklets, from twenty-four to twenty-six in number, and hence is called the armed tapeworm (Fig. 241).

The neck is narrow and thread-like, nearly 1 inch long. The body is divided into segments, which possess both male and female generative organs, and at about the four hundred and fiftieth they become mature and contain ripe ova. The segments are about 1 cm. in length and from 7 to 8 mm. wide. The worm attains its full growth in about three to three and a half months, about which time the segments are continuously shed and appear in the stool. The uterus forms a straight median tube in each segment, giving off five to seven branches on each side. The branches are undivided at first, but finally ramify as a tuft (Fig. 242).

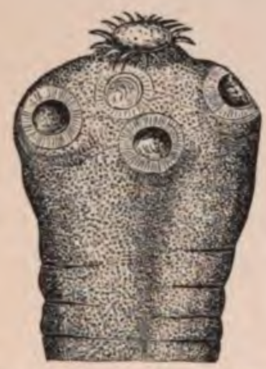

Fig. 241.—Head of Tænia solium, rostellum with hooks; suckers (Mosler and Peiper).

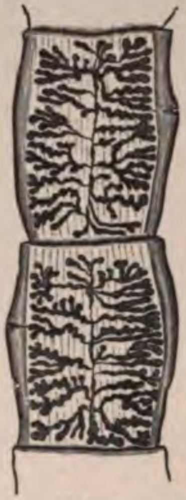

Fig. 242.—Mature segments of Tænia solium; proglottides; uterus and branches (Mosler and Peiper).

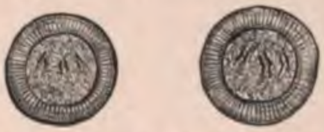

Fig. 243.—Eggs of Tænia solium, showing thick shell (Mosler and Peiper).

The eggs are rounded and provided with a thick shell (Fig. 243).

Rarely the cysticerci (measles) are found in man, as in the muscles, brain, eye, and skin.

In the muscular system they cause pain, numbness, weakness, and symptoms a little like peripheral neuritis. In the ventricles of the brain irritative symptoms may result. In one case diabetic symptoms were reported. They can be recognized in the eye. Tænia solium was formerly believed to exist alone, but several have been found together.

Tænia saginata, or Mediocanellata, the unarmed or beef tapeworm.

This is the most common form seen in America as well as abroad. It is longer, thicker, and wider than the Tænia solium. It may attain a length of 15 to 20 feet (approximately $4\frac{1}{2}$ to 6 meters) or more. The head measures over 2 mm. in breadth, has four large sucking disks, but no hooklets and no rostellum (Fig. 244). It is square shaped.

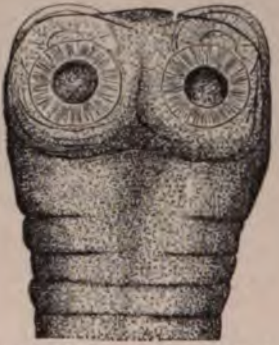

Fig. 244.—Scolex of Tænia saginata (Mosler and Peiper).

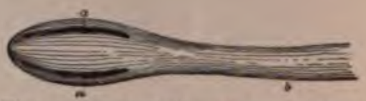

Fig. 246.—Bothriocephalus latus: *a*, *a*, Head; *b*, neck (Blanchard).

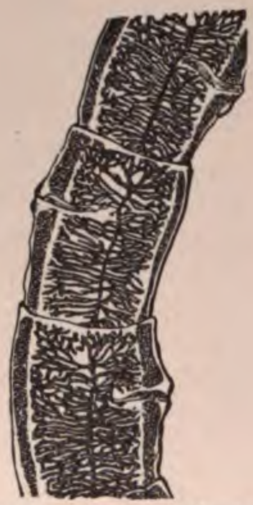

Fig. 245.—Segments of Tænia saginata (Mosler and Peiper).

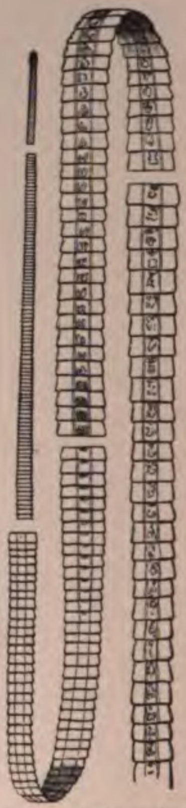

Fig. 247.—Bothriocephalus latus (Eichhorst).

The ripe segments are from 17 to 18 mm. in length and from 8 to 10 mm. in breadth. The uterus consists of a median stem, with from about twenty to thirty-five lateral branches (Fig. 245).

The ova are larger and the shell thicker, and possibly slightly more elliptic, but the two forms are difficult to distinguish by their ova. The measles (cysticerci) occur in beef and are smaller than those of the Tænia solium. Human beings acquire this worm by the consumption of raw beef.

Bothriocephalus Latus (Tænia Lata, or Pig Head).—This is found in certain districts bordering on the Baltic Sea, in Holland, Switzerland, and Japan. A few cases have occurred in the United States, believed generally to have been imported.

The parasite is large and long, measuring 25 to 30 feet or, more approximately, 7½ to 9 meters.

The head is elongated, almond shaped, being about 2 mm. long and 1 mm. broad; it has two grooves, probably suckers, on its flat surface (Fig. 246).

It has no hooklets. The neck is narrow and short, about 2 cm. in length, and passes at once into the body segment. The body is thin and flat (Fig. 247). The full-grown proglottides are nearly square and show the sexual organs in the center.

The uterus presents as a median dark line, with four to six lateral branches, looking like a star or rosette. The eggs are oval, round, with a thin membrane and a lid (Fig. 248). They measure 0.07 mm. long and 0.04 mm. in width. The larvæ develop in the peritoneum and muscles of pike especially, and of fish such as the turbot, perch, and trout. Infection occurs through eating insufficiently cooked fish.

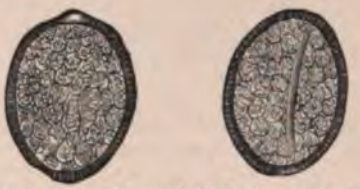

Fig. 248.—Eggs of Bothriocephalus latus (Mosler and Peiper).

Tænia lata occur in the small intestine of men and rarely in dogs. A severe and even fatal form of anemia may result from this worm.

There are a few rare forms of tapeworms occasionally found in human beings.

Tænia nana (Hymenolepsis nana) occurs chiefly in Italy, occasionally in Egypt. It is the smallest tapeworm found in man, measuring 10 to 15 mm. long, and may have nearly two hundred segments. The head has four sucking disks, a rostellum, and about twenty-four hooklets in a single row. Proglottides are short and broad. It is found more frequently in children, and occurs in large numbers in the small intestine.

Nervous disturbances, fainting spells, and even epileptiform attacks are produced thereby.

The *Davainea Madagascariensis* (Tænia Madagascariensis) is a rare form of this worm.

Tænia cucumerina (Elliptica, or Dipylidium Caninum).—This is small, of cucumber shape, occurs frequently in the intestines of the dog, and has been found in small children. The larvæ develop in the lice and flees of the dog. The worm is 10 to 40 cm. long and about 3 mm. wide.

Tænia Flavopunctata (Hymenolepsis Diminuta).—This has been met with in about 12 cases. The worm is 2 to 6 cm. long and about 3 mm. wide. Its head is small, club shaped, and provided with sucking disks. It is common in rats. The larvæ develop in Lepidoptera and in beetles.

Bothriocephalus Cordatus.—This tapeworm resembles the Bothriocephalus latus, except that it is shorter and the head merges into the proglottides without an intervening neck. It occurs in the intestines of men and dogs in Greenland.

Other types of tapeworms occur, but they are excessively rare and not found in Europe or America.

Echinococci are the larvæ of the Tænia echinococcus of the dog. The latter is a tiny cestode 4 or 5 mm. long, consisting of three or four segments, of which the terminal one alone is mature. The head is small, provided with four sucking disks, and a rostellum with a double row of hooklets.

As a result of the ingestion of these parasites, cysts develop in various parts of the human organism, as in the liver, muscles, etc. These cysts contain scolices, the head of the tænia presenting four sucking disks and a circle of hooklets.

Cysts have been passed per rectum. The disease is common to Iceland, not uncommon in Europe, but rare in this country. The reader is referred to Echinococcus Disease in any work on Practice of Medicine.

Treatment of Tapeworms.—To escape infection avoid raw, medium-done meats, pork, and fish. One should not trust to meat inspection alone. Thorough cooking of the meat is the only guarantee of extinction of the cysticerci. All worms or fragments removed should be destroyed by burning, and if one handles the proglottides or ova, the hands should immediately be disinfected; also the stools.

For about two days before administering a vermifuge the patient should be kept on a scanty diet, consisting of broths, soups or milk, with a few crackers. The night before, no food; and in the morning a cup of tea or coffee, followed in about an hour by the vermifuge.

Calomel, gr. 5 (0.3), castor oil, ℥ss to j (15.0–30.0), or a saline cathartic should be given daily for a couple of days previously.

Male fern is considered quite efficient.

Extract of filix mas, ethereal, ʒiss to iiss (6.0–10.0), mixed with simple syrup; follow in a couple of hours by a saline cathartic or castor oil.

The following have also been suggested: Oleoresin aspidii (male fern), ʒss to j (2.0–4.0), in capsules, coated with keratin; a few hours later castor oil, ℥j (30.0).

Filicic acid (filmaron), an amorphous principle from root of male fern, insoluble in water. It should not be administered in fatty oils or alcohol, as they dissolve it and it is toxic. Give in capsules, gr. viiss to xv (0.5–1.0); follow by a saline purgative (not castor oil).

Pomegranate root is efficient, given as an infusion of the *bark;* 3 ounces are macerated in 10 ounces of water and then reduced to one-half by evaporation. The entire quantity is taken in divided doses within an hour or more. It is effective, though sometimes producing colic.

The active principle of the root, *pelletierin*, gr. iv to viiss (0.25–0.5), in sweetened water, to which tannin, gr. v (0.3), can be added, may be given as a substitute. These remedies are followed in a couple of hours by a purge. Pelletierin tannate can be secured and given in the same dose.

Pumpkin seeds (Semina cucurbitæ), 3 or 4 ounces (90.0–125.0), should be bruised and macerated for twelve to fourteen hours; then mixed with a little grape-sugar, diluted with milk, Oj (500 cc.), and take in two doses about half an hour a part; then follow in two hours by castor oil.

Turpentine, Oleum terebinthinæ (spirits of turpentine), ℥j (30.0), in honey or with sugar, follow with a glass or two of milk; and two hours later a cathartic.

I have found *pine-needle oil* (Gardner's), ʒj to ij (4.0–8.0), also efficacious.

Cusso (kousso), cusso pulv., ℥ss (16.0); mel depuratum (honey), ℥ss (16.0).

Or cusso can be mixed with sugar, water, or lemonade, and taken in divided doses within an hour. Though cusso is cathartic, it is preferable to follow in two hours with a dose of castor oil.

Kamala, pulv. kamala, ʒj to ij (4.0–8.0), suspended in syrup or in wine. This preparation is purgative and may cause griping, nausea, and vomiting. The dose should be distributed over an hour. Castor oil may be given later.

Cocoanut has been recommended as a vermifuge; the milk and albumin of an entire nut should be taken within an hour.

Naphthalin, in capsules, gr. 10 to 30 (0.6–2.0), in divided doses, within a few hours.

Salol, gr. 45 (3.0), in capsules, in divided doses, has been recommended.

A combination of these remedies is often effective. Thus: take ℥ss (16.0) of an infusion of pomegranate seeds; pumpkin seeds, ℥j (30.0); pulv. ergot, ʒj (4.0), and boiling water 10 ounces (300 cc. approximately). Make an emulsion of male fern—ʒj (4.0) ethereal extract with acacia powder. Mix the emulsion and infusion and take fasting at 9 A. M. Follow a couple of hours later with castor oil or a saline cathartic.

Osler recommends the addition of croton oil, ♏ij (0.118), to the above, but I think this rather too active treatment. Male fern, pumpkin seed, pomegranate, and turpentine are the best remedies.

About two hours after the vermifuge a cathartic, such as citrate of magnesia, ʒj to ij (4.0–8.0), or magnesium sulphate, larger doses, or some other saline cathartic, or castor oil, ℥j to ij (30.0–60.0), should be given.

The head of the tapeworm should be looked for, as the parasite will regrow if this is not removed. In some cases this is difficult to find. Children require proportionately smaller doses, according to their age. Care must be exercised with patients who are debilitated, recently convalescent from typhoid, or have severe intestinal disorders. It may be necessary to postpone treatment.

Trematodes (Fluke Worms); Distomiasis.—Flukes are *found in the lungs, liver, small intestine, and in the blood;* in the latter case affecting chiefly the urinary system and the rectum.

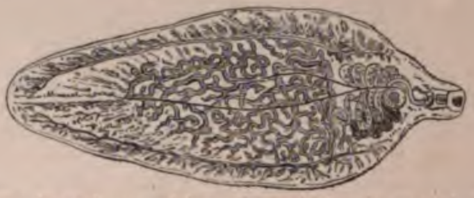

Fig. 249.—Distoma hepaticum, with male and female sexual apparatus; × 2½ (Leuckart).

The trematodes are solid worms of leaf or tongue shape. They possess a clinging apparatus in the form of oral and ventral sucking cups, which vary in number. Sometimes they also have hook or clasp-like projections. The intestinal canal is without an anus and is split like a fork. They are generally hermaphroditic.

Flukes are found in China, Japan, India, Egypt, Arabia, and Persia, and imported cases have been found in Canada and the United States.

They have been found in the cat, dog, and hog in this country.

Five species of *liver fluke* are known to occur in man. The Distoma conjunctum, the Indian liver fluke, usually described, the Distoma lanceatum (lancet fluke), and the Distoma sinensis are the most frequent, occurring in the liver, the last being most important.

The Fasciola hepatica, common to ruminants, and the Opisthorchis (Distoma) felineus, occurring in Prussia and Siberia, and found in cats in Nebraska, are the other varieties.

In general, we may say the liver fluke is of leaf shape. It may vary in length from 10 to 20 mm. by 2 to 5 to 10 mm. broad. The cephalic end projects like a beak and has a small cup-like sucker, in which lies the mouth. Behind this, on the ventral surface, is a second cup; and between the two is a special orifice. The uterus appears as a convoluted bag behind the posterior sucker. On each side of the body are the ovisacs and the branched testicular canals (Fig. 249).

The eggs are oval, 25 to 30μ long by 15 to 17μ broad, of brown color, with a sharply defined operculum (lid) (Fig. 250).

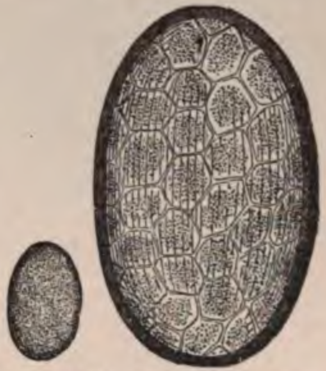

Fig. 250.—Eggs of Distoma hepaticum and Distoma lanceolatum, moderately magnified (Heller).

The *Distoma lanceolatum*, another variety of liver fluke, has a lancet shape and the head is not especially marked off from the body (Fig. 251). The eggs are rather small, 0.04 mm. long (Fig. 252).

Young children suffer more frequently from liver fluke; sometimes whole families or villages are attacked.

There is an irregular diarrhea; there *may or may not be blood*. The liver enlarges. There is often pain and an intermittent jaundice, but not much fever. Anasarca and ascites come on later. The ova of the parasite are found in the stool.

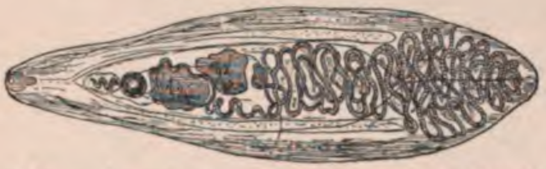

Fig. 251.—Distoma lanceolatum with its inner organs; × 10 (Leuckart).

Intestinal Distomiasis.—In India the Distoma fasciolopsis has been found in a number of cases in the small intestine, with diarrheal symptoms.

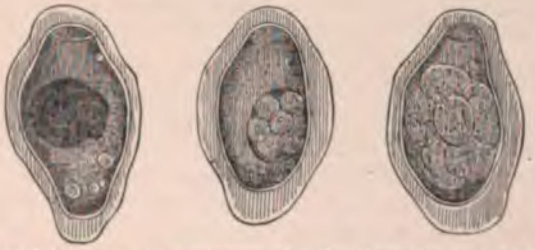

Fig. 252.—Egg of Distoma lanceolatum shortly after the formation of a shell; × 400 (Leuckart).

Hemic Distomiasis; Distoma Hæmatobium; Bilharziasis; Schistosoma Hæmatobium; Bilharzia Hæmatobia.—Endemic hematuria, particularly in Egypt, had been known for many years, when in 1851 Bilharz discovered the parasite of the disease. The blood fluke prevails in South Africa (the Transvaal); in North Africa, *especially in Egypt;* in Arabia, Persia, and the west coast of India. It is prevalent in Japan. It has been observed in Porto Rico and in the Philippines. The disease is rare in the United States, only 7 cases having been reported. In addition, 7 cases occurred among the Boers, who were on exhibition in this country after the African War.

The first case of rectal infection reported in the United States was in a German, who evidently contracted the disease in Brazil and who was treated at the German Hospital. He suffered from mixed infection—Strongyloides intestinalis, Trichocephalus, and Schistosoma hæmatobium, reported by L. Blumgart,[1] of New York.

The Schistosoma has separate sexes and carries the female in a gynecophorous canal. The male is from 12 to 14 mm. long. Its body has ciliated warts on the integument, but otherwise is smooth, and in the posterior portion is rolled up into a tube, which serves for the reception of the female (Fig. 253).

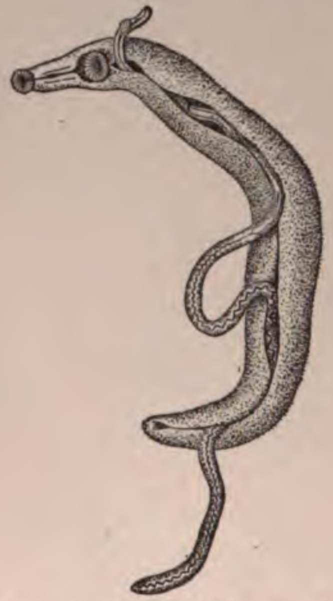

Fig. 253.—Male and female of Bilharzia hæmatobia (Loos).

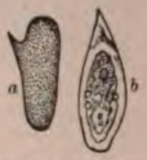

Fig. 254.—Eggs of Distomum hæmatobium (Bilharzia hæmatobia), length, 0.12 mm.; breadth, 0.05 mm.: *a*, Egg with lateral spine; *b*, egg with terminal spine; × 150 (after Bilharz).

There is a sucker at the anterior end and a second one posterior to it on the ventral surface. The female is from 16 to 18 mm. long and nearly cylindric. The eggs have a terminal or lateral spine (Fig. 254). The trematode is most abundant in the blood of the portal system, while the ova lodge in the capillaries, especially of the bladder, urinary organs, rectum, and lungs. In the stools of the case reported, the spine was placed laterally on the ova.

Infection is now considered to take place in two ways—either by the gastro-intestinal tract, through infected food or water, or through the skin by bathing in infected streams.

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[1] Medical Record, April 6, 1907.

The parasite reaches the portal system, where it develops. The males, bearing the females, creep to various parts, particularly the bladder and rectum. The eggs are laid in the tissues, but wander like other sharp foreign bodies, and escape with the urine and feces. The eggs in the tissues cause irritation, fibroid changes, and papillomata in the bladder and rectum. Hematuria and bladder irritation, chronic cystitis, tenesmus, mucus and blood in the stools, ulcerative proctitis, calculi in the kidney and bladder, peri-urethral abscess and perineal fistula, vaginitis, inflammation of the ureters and seminal vesicles, may all occur. Bilharzial colitis has been reported.

Anemia is present and eosinophilia is quite marked.

In 1904 there was described a new blood fluke, Schistosoma Cattoi, or Japanicum, found in Japan. It lives chiefly in the vessels of the alimentary canal and ulcerative lesions are found therein. The ova appear in the feces. Katsurada has studied numerous cases. This condition is known in Japan as the "Katayama disease," from the name of a town in which it is quite prevalent.

Catto described certain bodies he found in a Chinaman in 1904, and the new fluke is sometimes called by his name.

Paul G. Woolley[1] has reported a case occurring in the Philippines, and has given an excellent description of the disease.

The worms are characterized by the *absence of ciliated warts on the integument,* which *are a feature of the Schistosoma hæmatobium.* The worm averages 10.43 mm. long. The eggs are smaller, brown in color, have blunter ends, and *no spine.*

Katsurada gives the most definite reports of the disease "Katayama," according to Woolley:

"Defective physical development is the rule in the affected children. Diarrhea is usually the first symptom to be noted, while anemia and ascites generally follow later; the most striking feature is the shape assumed by the trunk. The hypogastric region seems to shrink, while the epigastric enlarges, a transverse furrow forming directly above the umbilicus, so that the general appearance of the abdominal region is that of an inverted gourd. Dilatation of the epigastric region and of the lower part of the thorax were noted even in patients whose liver and spleen were not much enlarged. The commonest symptoms are an initial increase in the size of the liver, followed by a decrease, a secondary enlargement of the spleen, a mucosanguineous diarrhea, severe attacks of ascites, and progressive anemia." Katsurada found the ova of the parasite under discussion, and also those of Trichocephalus dispar, Uncinaria, and Ascaris lumbricoides in the stools of his patients.

The rectum and appendix were the parts chiefly affected, but the ova were found in the subperitoneal layer, the submucosa, and mucosa, especially in necrotic areas, from the cecum to the anus. Adult trematodes were found in the blood-vessels.

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[1] Philippine Journal of Science, Jan., 1906, vol. i, No. 1.

In Woolley's case there were also amebæ and the ova of the uncinaria; but in the fibroid tissue of the submucosa of the large intestine there were many ova of the Schistosoma Japanicum surrounded by round-cell infiltration (Fig. 255). A type of cirrhosis was produced in the liver. Splenomegaly, ascites, dysentery (specific), and possibly Jacksonian epilepsy may be produced by these trematodes, according to Woolley.

The disease is probably water-borne, originating in rice-fields or irrigated gardens, from human fertilizer. Infection may occur through the skin or by the gastro-intestinal canal.

Treatment.—The extract of the male fern is recommended for distomiasis and the treatment as of tapeworm. Nothing has been found for the treatment of the parasite in the blood. The author would suggest the trial of urotropin, gr. 10 (0.6), and sodium benzoate, gr. 10 (0.6), three or four times a day. The latter lessens the irritation of the urotropin, as demonstrated by William H. Thomson.

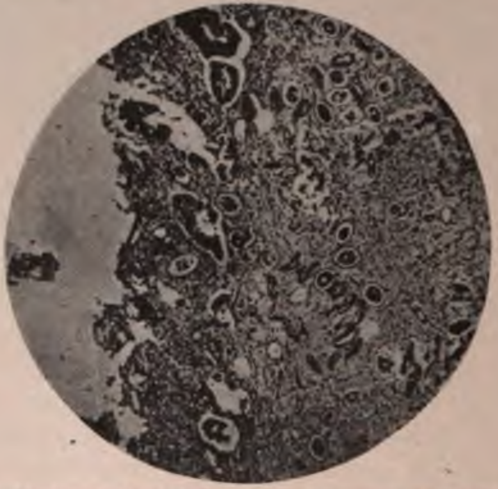

Fig. 255.—Schistosoma Japanicum. Ova in mucosa and submucosa of large intestine. Shows atrophic and infiltrated condition of mucosa. Hematoxylin (Woolley).

The inflammation of the bladder, colon, and rectum should be treated as indicated by irrigation, etc. Solutions, as described under Proctitis and Colitis, can be employed for the latter complications.

Nematodes (Round Worms).—Round worms have a slender, cylindric, at times a filiform body, with neither segments nor appendages. The integument is thick and elastic. The mouth is at one extremity and furnished with either soft or horn-like lips. The alimentary canal extends through the entire body and terminates in an opening on the ventral side near the posterior extremity. The sexual organs and their orifices are on the ventral surface. The female aperture is at the middle of the body, while in the male the sexual orifice is near the anus. The males are usually smaller than the females.

Ascaris Lumbricoides (Round or Spool Worm).—This is one of the common parasites observed in man. It is cylindric in shape, pointed at both ends, and of a yellowish-brown or slightly reddish color. It varies from 4 to 12 inches (10–30 cm.) in length, the female being as large as 12 inches (30 cm.), while the male is only one-half or two-thirds the length, 8 inches (20 cm.), and frequently much smaller.

The posterior extremity of the male is bent in the shape of a hook and furnished with two spicules or chitinous processes (Fig. 256). The mouth has three muscular lips provided with very fine teeth.

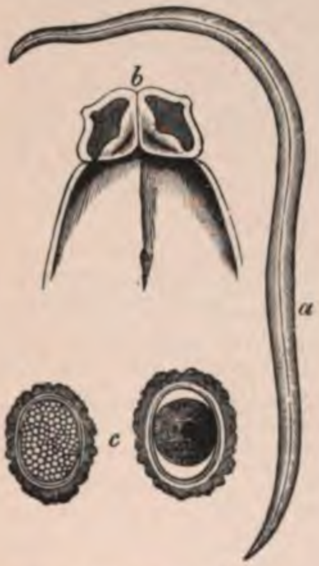

Fig. 256.—Ascaris lumbricoides: *a*, Body; *b*, head; *c*, eggs (after v. Jaksch).

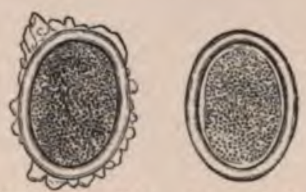

Fig. 257.—Eggs of Ascaris lumbricoides, double shell; albuminous envelope, magnified (Mosler and Peiper).

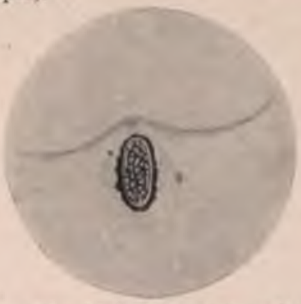

Fig. 258.—Unfertilized egg of Ascaris lumbricoides; ×500 (Logan).

The worm is transversely striated and has four *longitudinal bands.*

The sexual opening of the female is anterior to the middle of the body. The eggs when ripe have a double shell, and around this is an albuminous envelope, irregular in shape, studded with excrescences (Fig. 257). The long diameter of the ovum is about 0.075 mm. and 0.058 mm. in width. Atypic (unfertilized) eggs have been described by O. T. Logan[1] of China. When taken from the uterus it has not the typic thick shell, but is granular, elliptic, and enclosed in a delicate membrane (Fig. 258).

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[1] New York Medical Journal, Dec. 21, 1907.

In the feces, the yelk is not finely granular, but coarsely globular; the albuminous coat is less voluminous and projects from the shell like blunt saw teeth. The unfertilized egg[1] is longer and narrower than the fertilized egg and markedly elliptic, with a tendency to flatten at one or both ends. It is occasionally oval, but never round.

An irritating, odoriferous substance is formed by the round worm. Huber[2] states that it may occasion urticaria in those predisposed to this symptom. Peiper suggests that the nervous symptoms, sometimes resembling meningitis, may be due to this poison; and Chauffard and Marie report fever, intestinal symptoms, diarrhea of intermittent character and foul breath, so-called typholumbricosis, in connection with these worms. The fever may continue for a month or more.

The parasitic life history is direct, by ingestion of the ova, with no intermediate host. The parasite occupies the upper part of the small intestine. Usually not more than one or two are present, but they may occur in enormous numbers.

Infection usually takes place by eggs in the soil near dwellings, in the drinking-water, and especially in raw foods, such as salads and fruits. These worms occur more frequentiy in children from three to twelve years of age and in the poorer class. They are not so frequent in adults. Females seem more frequently infected.

Migration.—The worms may crawl into the stomach, whence they may be ejected by vomiting; or they may pass through the esophagus and enter the pharynx, whence they may be withdrawn. The worm has entered the larynx and has produced fatal asphyxia, or into the trachea and lungs and caused gangrene. They have passed through the Eustachian tube and appeared at the external meatus. They have been found in the bile-ducts, the gall-bladder, and even in the liver, where they produced fatal abscess. They have entered hernial sacs, perforated intestinal ulcer, and some claim even the healthy bowel wall has been perforated by them. Appendicitis has been attributed to the ascaris; and obstruction of the bowel is said to have been produced by a large mass of ascarides.

Symptoms.—They may produce no symptoms. In children, irritability, restlessness, picking at the nose, grinding the teeth, twitchings or convulsions, have been attributed to them. Anorexia, nausea, irregular bowel action, meteorism, irregular pulse, and black rings around the eyes may also occur. In rare instances progressive anemia has been observed. Itching of the nose may be present. The worms probably produce local hyperemia of the intestinal walls.

Diagnosis.—This is made by the detection of the worm or of its ova in the stools.

Treatment.—The stools should be disinfected by carbolic (5 per cent.) or bichlorid (1:1000) solution to destroy the ova. The hands should be disinfected and all food protected against infection.

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[1] New York Medical Journal, Aug. 19, 1905 (Wellman); Reference Handbook Medical Sciences, p. 502.

[2] Twentieth Century Practice of Medicine, vol. viii, p. 583.

It is preferable to administer a simple saline for a couple of days and keep the patient on a light diet before administering the anthelmintic.

Santonin is the best remedy. It can be given mixed with sugar in doses of gr. $\frac{1}{3}$ to $\frac{1}{2}$ (0.022–0.032) for a child, and gr. 2 to 3 (0.13–0.194) for an adult, followed by calomel or a saline purge.

It can be administered in divided doses; thus, santonin, gr. 1 (0.06), three or four times a day, followed by a purge; or santonin, gr. $3\frac{1}{2}$ (0.2), with castor oil, ℥ij (60.0); give 1 teaspoonful for a small child; 1 dessertspoonful for a larger child; 1 tablespoonful for an adult, two or three times daily (Einhorn).

Santonin, gr. $\frac{1}{3}$ (0.022); hydrargyrum choridum mite, gr. 1 to 2 (0.065–0.13). Give one powder t. i. d. Yellow vision (xanthopsia) occasionally follows the use of santonin.

Chenopodium (powdered seeds), in doses of gr. xv to xxx (1.0–2.0), or oleum chenopodii, ♏ij to x (0.118–0.592), followed by a cathartic, have been employed.

Thymol, gr. viiss to xxx (0.5–2.0), given in capsules in divided doses and followed by a saline cathartic, has been recommended.

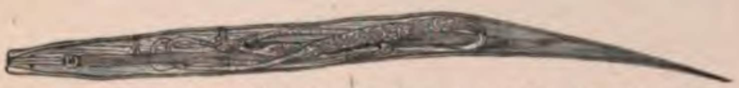


Fig. 259.—Oxyuris vermicularis: Female, enlarged (Mosler and Peiper).

Enteroclysis with water, to which a few drops of benzine are added, has been suggested; but I scarcely see the benefit, as the habitat of the worms is in the small intestine.

Ascaris Mystax.—This is a round worm resembling the ascaris lumbricoides, but smaller and thinner. It is found chiefly in cats and rarely in man. No special symptoms result.

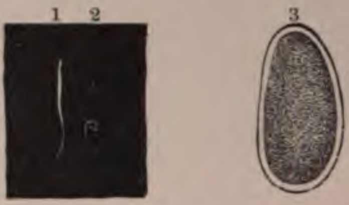
Oxyuris Vermicularis (Thread-worm; Pin-worm; Seat-worm; Awl-tail; Maggot-worm).—This parasite occupies the rectum and colon. It is white and filiform.

The male measures about 4 mm. ($\frac{1}{6}$ inch) in length and the female about 10 mm. ($\frac{2}{5}$ inch) (Fig. 259). It has three small nob-like lips. The female has two uteri, passing backward and forward from the end of the vagina. The opening of the latter is situated above the middle of the body. The eggs are 0.05 mm. long and about 0.02 mm. wide, with granular contents and white shell (Fig. 260).

Huber and others claim that they are generally deposited outside the body, so that feces rarely contain them; while Osler holds that they are usually found in the feces in large numbers. These worms occur at any age, though most commonly in children. The worms are found in the feces, at the anus, or in the vagina.

Infection takes place through drinking-water or through salads, radishes, fruits, etc., the ova being dried upon them, or through the unwashed hands of the host.

The eggs of the oxyuris reach the stomach, when the shell opens and the embryo migrates into the small intestine. After fructification the females pass along the canal to the cecum, where they remain until the eggs are ripe, and then pass on downward, chiefly to the sigmoid and rectum. The oxyuris may traverse the intestinal wall and have been found in the peritoneal cavity, where they may form verminous tubercles in Douglas' fossa or perirectal abscesses.



1, Female; 2, males; 3, ovum, magnified.
Fig. 260.—Oxyuris vermicularis, natural size (Vierordt).

Symptoms.—The oxyuris produce great irritation and itching around the anus, particularly at night. The pruritis ani is pronounced. The patient becomes nervous and irritable, sleep is disturbed. There may be anorexia, nausea, dizziness, palpitation, pollutions in the male, occasionally diarrhea, and at times anemia. Sometimes the parasites enter the vagina and cause irritation or nymphomania.

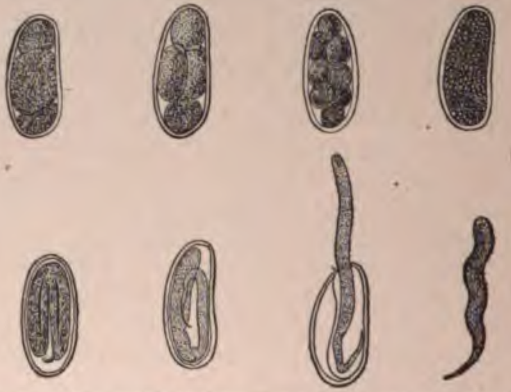


Fig. 261.—Segmentation and development of embryo of Oxyuris vermicularis (Heller).

Diagnosis.—The worms are easily detected in the feces and are readily diagnosed by their appearance and location through inspection of the anus.

Treatment.—Cleanliness of the hands of the infected person and disinfection of stool, clothes, and bedclothes are important. Sleeping with an infected case should be forbidden.

Raw fruits should be cleaned and peeled, salads, etc., thoroughly washed. One should not eat out of the same vessels as the patient.

Santonin, administered by the same method as for ascaris, is an excellent remedy. Local treatment by enemata of water, 1 quart (liter) containing ♏v to x (0.296–0.592) of benzine, or vinegar ℥iss to ij (45.0–60.0), or thymol (1 : 2500) by enema, or fluidextract of quassia, ♏x to xxx (0.292–1.704) to the quart; or soak a quassia cup in water for half an hour and inject quassia water, Oj (500 cc.).

An enema of spirits (oil) of turpentine, ʒj (4.0) to Oj (500 cc.) of water, is of value.

These injections should be given with hips elevated and retained for a short period. Cold injections of strong salt water are of service in children. Carbolic acid I believe unsafe. Injections of lime-water are of use.

Black wash—calomel, ʒj (4.0); lime-water, Oj (500 cc.)—locally, externally; lead-and-opium lotion, unguentum belladonna, vaselin, or

℞. Unguent. belladonnæ........................ʒij (8.0)
Tr. aconite radix..............................ʒss (2.0)
Zinc oxid..gr. xv (1.0)
Unguent. aq. rosæ........................q. s. ℥j (30.0).—M.
Sig.—Ft. ung. External use to anus.

Cocain, gr. ss (0.32), can be added to this. These preparations lessen itching.

The saline enemata, given frequently, are of use.

Ankylostoma (Anchylostoma) Duodenale; Uncinariasis; Hookworm Disease; Miner's Anemia; Egyptian Chlorosis; Dochmius Duodenalis or Strongylus Duodenalis.—In 1843 Dubini first described this parasite in man. Griesinger, in 1854, demonstrated it as the cause of Egyptian chlorosis. Subsequently it was described in the tunnel workers of St. Gothard, and it is now recognized as an important cause of tropical anemia and of the anemia of miners, brick workers, and tunnel workers.

Incidence.—This parasite is widely spread in tropical and subtropical countries, and is one of the most fatal of parasitic diseases. In Porto Rico, in 1903, 5736 deaths out of a total of 23,433 were from anemia due to uncinariasis, as shown by the Anemia Commission in the report issued by Ashford King and Igaravidez. Stiles has demonstrated that the disease is endemic in many places, and is the cause of the common anemia in the Southern States.

Bass[1] has reported a large number of cases in the country population of Mississippi. Uncinariasis has been found among the miners in Pennsylvania. It is not uncommon in the Philippines. The disease is prevalent among the miners of Germany and Austro-Hungary and also in Westphalia. The anemia of the Cornish miners has been shown to be due to hookworm. In Egypt the disease is very prevalent.

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[1] Journal American Medical Association, July 21, 1906.

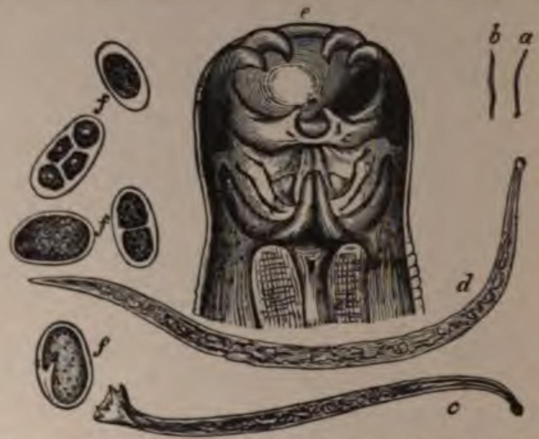

Fig. 262.—Ankylostomum duodenale: *a*, Male (natural size); *b*, female (natural size); *c*, male (enlarged); *d*, female (enlarged); *e*, head; *f*, eggs (after v. Jaksch).

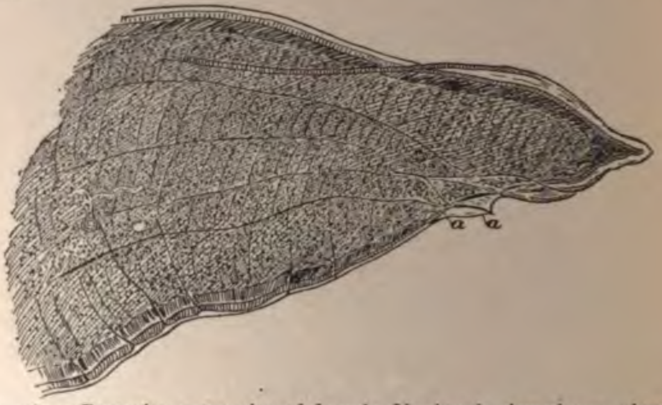

Fig. 263.—Posterior extremity of female *Uncinaria Americana*, viewed ventrolaterally, showing anal opening expanded: *a*, *a*, Anal papillæ, showing small chitinous tips (A. J. Smith).

Parasite.—The worm is a strongyle, closely allied to the sclerostoma, which causes verminous aneurysms and colic in the horse, and to the gapeworm of fowls. There are two forms—the Anchylostoma duodenale and the Uncinaria Americana—described by Stiles. They have the same general characteristics, there being certain differences in the arrangement of the teeth, etc.

The worm is cylindric in shape, about 0.5 to 1 mm. thick. The es are from 7 to 11 mm. in length, the females 10 to 18 mm. The erican worm is the longer. The worm is yellowish or grayish e in color, with translucent edges. The head is curved toward the al surface and the mouth is provided with a heavy armature of k-like teeth, with which they pierce the mucosa. There is a strong cular esophagus. The male has a prominent caudal expansion or sa (Fig. 262).

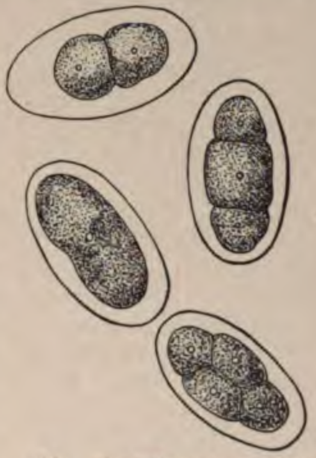

Fig. 264.—Four eggs of the New World hook-worm (*Uncinaria Americana*), in the one-, two-, and four-cell stages. The egg showing three cells is a lateral view of a four-cell stage. These eggs are found in the feces of patients, and give a positive diagnosis of infection. Greatly enlarged (after Stiles).

In the female the caudal end is ted and armed with an awl-like ng (Fig. 263). The eggs are oval, to 76μ long by 36μ wide (Fig. 264) he American form. They are laid egmentation. The development is ct, without an intermediate host. European eggs are smaller.

The embryo lives in the water or t ground and passes through the ditiform stage. Larvæ may live months in the mud and water of mines. They may be taken into body by drinking-water, with the from the hands of the miners and el workers, or in the soil eaten by arth feeders, the geophagi. They be carried in the dust and connate green vegetables and fruit.

Uncinarial Dermatitis.—Ashford King[1] refer to the fact that in o Rico infections through the th are rare, and that fully 96 cent. of the patients have suffered from ground-itch ("Mazam- "), *due to invasion of the skin[2] by these larvæ. The well shod were affected.* Various stages of dermatitis occur, and even obsti- ulcers of the leg, in the lower third especially. These were called pical" or "syphilitic" by the ignorant. Loos demonstrated that ryo worms enter the skin and are carried by the veins to the right of the heart and lungs, and then pass up through the trachea the pharynx and are swallowed. Ashford further believes that infections can take place without manifest dermatitis from riments on animals. Skin infection is probably very frequent. ction is more common in summer than winter, in part prob- due to the fact that greater protection is afforded the lower s during the cold or rainy season.

Uncinariasis, Journal American Medical Association, Aug. 10, 1907.

The skin eruption known as "bunches," occurring in the Cornish miners obably due to the entrance of these worms (Haldane).

Immunity.—Some whites, and *especially the negro race* and Asiatics, enjoy considerable immunity to this disease. One may find a large number of ova in the stools, and yet there may be few or no symptoms. Usually the greater the infection, the more acute the onset and course of the disease. The *adult worm lives in the small intestine;* more *are found in the jejunum;* many in the duodenum; and rarely in the ileum or colon.

Symptoms.—In the early stage there may be gastro-intestinal symptoms, such as pains in the epigastrium (gastralgia), tenderness in the right hypochondrium, nausea, occasionally vomiting, constipation, rarely diarrhea. Pain in the sternum and chest, slight breathlessness on exertion, and palpitation. A temperature of 37.5° to 38.5° C. (99.5°–103.5 F.) is not uncommon. The patient feels unable to work; gradually anemia becomes manifest, the fever disappears, hemoglobin steadily diminishes. Headache, vertigo, tinnitus aurium, hemic murmurs, weakening of the pulse, and dyspnea follow. The pains in the sternum become quite severe, debility and mental hebetude increase; patellar reflexes are diminished or even lost. Impotence or amenorrhea may be present.

In this advanced condition the skin is of a dirty muddy hue, at times waxy white. In the Southern States it is known as the "Florida complexion." The eyes are dull, heavy, lack lustre, and have a blank stare. Children are interfered with in their growth and become stunted and ill developed. The circulatory system becomes more profoundly affected; the heart, at first slightly hypertrophied, now becomes dilated and broken compensation ensues. There are edema of the feet and legs, puffiness of the face and a general anasarca, with pericardial, pleural, and peritoneal effusions. The patient is bedridden and gradually passes away in a semisomnolent condition. Acute cardiac dilatation, cerebral effusion, or violent diarrhea may be terminal events.

Osler holds that the liver and spleen become enlarged, but this does not correspond to the findings of Ashford and King. The hemoglobin may fall below 30 per cent. and sometimes even to 8 per cent., and some of these latter have recovered under appropriate treatment.

Morbid Anatomy.—*Autopsies* (Ashford and King).—Muscles are often brownish gray, friable, and atrophied. Skin and subcutaneous tissue pale and sodden with fluid. Serous effusion generally present in the pleuræ and pericardium, sometimes in the cerebral ventricles. Ascites marked.

Lungs.—Edema and passive congestion.

Liver.—Never normal; once was increased in size; never diminished; fatty degeneration often present; connective-tissue increase is not a feature.

Kidneys.—Chronic parenchymatous or chronic diffuse nephritis. As a rule, there was little connective-tissue increase.

Stomach.—Uncinariæ are occasionally found in the stomach, even adherent to its walls. Chronic gastritis is common.

Intestines.—The jejunum contained most of the uncinariæ, some unattached and others attached to the mucosa. The balance were mostly found in the first part of the duodenum, a few in the ileum, and none in the colon.

The intestinal lesion is confined to the mucosa, there *being a tiny superficial erosion* about 0.5 mm. ($\frac{1}{50}$ inch) in diameter, and not a deep ulcer. They are usually not red and are difficult to find, except with a hand lens. The duodenum and especially the jejunum are the seat of a chronic intestinal catarrh.

The dejecta are often of brownish color. Microscopically, eggs of the parasites and at times Charcot-Leyden's crystals are found in the stools.

Spleen.—This is frequently found reduced in size, soft, and has a wrinkled capsule. There was a paucity of lymphoid elements.

Hemolymph Glands.—In the region of the abdominal aorta, especially near the bifurcation, enlarged glands of dull reddish hue were noted. There was no surrounding trace of imflammation. Microscopic examination showed they were hemolymph glands of the type described as splenolymph.

Bone-marrow.—Changes such as occur in pernicious anemia; also eosinophilous cells.

Anemia.—Blood.—The findings vary from a diminution of hemoglobin and red cells to those of *pernicious anemia.* The hemoglobin is usually relatively lower in uncinariasis; as low as 15 to 20 per cent. in severe cases is not uncommon, 8 per cent. having been registered.

Polychromatophilia, and in severe cases poikilocytosis, with macrocytes and microcytes, occur. Normoblasts and megaloblasts were not uncommon, but the latter were in the minority. Erythrocytes averaged 2,406,416 per cubic millimeter, the lowest 754,000; average hemoglobin slightly over 40 per cent.

Osler reports marked leukocytosis in his cases. Some observers hold that leukocytosis is generally due to complications; while Ashford and King find no constant leukocytosis, but that it is present at times in acute cases, while in chronic cases there is apt to be leukopenia. There seemed to be a tendency of the leukocytes to degenerate.

Eosinophilia is of importance. Boycott and Haldane hold it is present in 94 per cent. of cases, and at times it is quite high. Ashford and King call especial attention to the fact *that in the most serious cases it is liable to be absent,* and that it is chiefly of prognostic importance. Very chronic cases of severe type, poor resisting power, and lack of blood regeneration, they state, rarely show eosinophilia, or only to a slight degree; that a rise of eosinophilia is of good prognostic significance; that a fall, with lack of improvement in the symptoms, is not a good omen. They hold that good resistance to the toxin of uncinariæ is expressed by eosinophilia.

Diagnosis.—By the finding of the eggs and parasites in the stool. It is well to examine the stools after a dose of thymol, followed by a saline cathartic. Stiles suggests placing a small bit of feces on a white blotting paper when a microscopic examination cannot be made. In about an hour there is a blood-red or reddish-brown stain suggestive of blood. The worms to the naked eye are about ½ inch long, of the diameter of a pin, with one end sharply recurved.

Eosinophilia is suggestive. The microscopic examination of the stool will settle the diagnosis.

Prognosis.—This is good if the condition is detected early, but is bad in the advanced cases.

Prophylaxis.—In camps or mines, proper sanitary regulations, such as the correct location of the latrines, etc., are important. In regions where this infection is endemic, new miners should be inspected, and infected cases should be pronounced free from the disease before being allowed to resume work. The feet and legs should be protected from the soil (should not be bare) and the hands properly scrubbed before eating. The stools of infected patients should be disinfected with bichlorid of mercury (1:1000) or carbolic acid (1:20) solution.

Raw fruits and vegetables, such as apples, lettuce, etc., should be properly cleaned, and should preferably be avoided if there are many cases of ankylostomiasis.

Treatment.—Filix mas, thymol, and betanaphtol are the best drugs. The ethereal extract of filix mas, same dosage as for tapeworms, and then the solid extract were tried in Porto Rico, but they seemed to be of no value. Ashford believes that possibly the preparation deteriorates in warm climates.

Thymol proved to be the best remedy. The day previous the patient abstains from solid food and at night is given 25.0 gm. sodium sulphate or any good saline cathartic; the next morning, if free bowel action, on an average about gr. 30 (2.0) of thymol should be given, and an equal dose two hours later, followed in a couple of hours by another saline cathartic.

This procedure should be kept up once a week, until no ova are found. Smaller, occasionally even larger, or more frequent doses may be required. It is generally advised that oily cathartics, such as castor oil and also alcohol, should not be taken directly after the thymol. Dizziness or slight collapse may follow the use of thymol. Ashford mentions cases who have taken alcohol for these conditions with no deleterious effect.

Betanaphtol, gr. xv (1.0), and two hours later the same dosage, administered like thymol, have been employed. It is somewhat irritating to the kidneys; is an excellent anthelmintic, but not quite as safe as thymol nor as efficacious.

Ashford and King hold that hemoglobin increased more rapidly under the use of iron, but that the return to normal came about as surely without it, after the use of thymol. They used Blaud's pills in obstinate cases. Iron and arsenic I believe valuable accessories.

℞. Blaud's pill (iron) (made fresh).............gr. v (0.3)
Sod. arsen..............................gr. $\frac{1}{50}$ (0.0013).—M.
One pill.
Sig.—One t. i. d.

Peptomangan (Gude), ℥j to iij (4.0–12.0), or Blaud's iron pill, gr. v to x (0.3–0.6), or iron tropon can be used, combined with Fowler's solution of arsenic, ♏v (0.274) t. i. d.; or atoxyl, gr. $\frac{1}{3}$ (0.02), by hypodermic every other day.

A subsequent high enema of thymol, 1 quart (liter), 1:2500, might be of value in aiding their destruction after the worms have passed into the large intestine.

Strongyloides Intestinalis.—Under this name we now include the small nematode worms found in the feces and formerly described as Anguillata stercoralis, Anguillata intestinalis, and Rhabdonema intestinale. The parasite occurs abundantly in the stools of the endemic diarrhea of hot countries, and has been described by the French in the diarrhea of Cochin-China. It has been found in Manila by Strong.

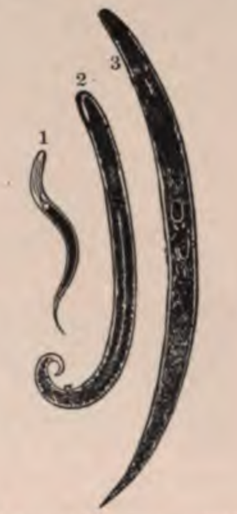

Fig. 265.—Strongyloides intestinalis and stercoralis: 1, Larva (Anguillula intestinalis); 2, male Anguillula stercoralis; 3, female Anguillula stercoralis (after Perroncito).

W. S. Thayer reported 3 cases from Osler's clinic. It has occurred in Italy.

Blumgart[1] reports their larvæ in a case and refers to reports of 5 additional cases, and to the fact that Southern physicians refer to other cases, so that the disease is probably more widespread than is supposed. The worms are said to occupy all parts of the intestines, and have even been found in the biliary and pancreatic ducts.

The female is from 1 to 2.20 mm. long and 0.04 to 0.03 mm. wide; the male is about a fifth smaller (Fig. 265). The mouth has three distinct lips, continuous with a triangular esophagus, which after narrowing dilates again into a second ovoid enlargement, which is followed by intestines. The intestinal tract is bordered by fine granulations.

Eggs are elliptic, with a thin, clear yellow shell, with granular contents about 0.00675 by 0.0375 mm. They hatch quickly, so are rare in the stools. They occur chiefly in the duodenum and jejunum, but have been found in the stomach and other parts of the intestines.

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[1] Medical Record, April 6, 1907.

Symptoms.—They are usually more of chronic diarrhea than of dysentery. There are at first mild dyspeptic symptoms, eructations, anorexia, and a diarrhea of moderate intensity, with soft and pasty stools, three or four a day, often in the early morning hours. The attacks are sometimes dysenteric, with mucus and blood; in other cases they are more choleraic, the dejecta consisting of liquid yellow material; while vomiting, cyanosis, and collapse may occur.

Emaciation and prostration may be present. Anemia is, as a rule, not very severe. Intercurrent dysentery is not uncommon, also headache, vertigo, tinnitus aurium, and prostration.

Treatment.—Rest and liquid diet. Male fern, ethereal extract, 12 to 30 gm., divided in three doses during the morning and repeated daily, have been used by the Italians.

Thymol has been quite successful.

Large quantities of olive oil have seemed to give good results in some cases.

Trichocephalus Dispar (Whip-worm).—This parasite is found in the cecum and large intestine of man. It measures from 4 to 5 cm. long, the male being smaller than the female. It is readily recognized by the peculiar differences between the anterior and posterior portions. The anterior forms three-fifths of the body, is thin and hair-like; the tail end of the female is more conic and thicker, terminating in a blunt extremity; while that of the male is rolled like a spring (Fig. 266).

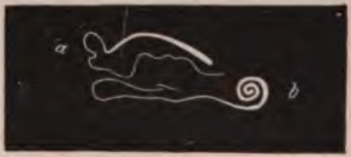

Fig. 266.—Trichocephalus dispar: *a*, Female; *b*, male (natural size) (Heller).

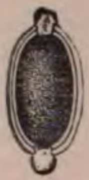

Fig. 267.—Egg of Trichocephalus dispar, moderately enlarged (Heller).

The ovum is lemon shaped, dark brown, 0.05 mm. in length, and provided with button-like projections (Fig. 267).

The number of worms is variable, as many as a thousand having been counted; often only ten to twenty are found. In parts of Europe they are very common, but not so in the United States. Profound anemia with diarrhea have occurred from them. Many worms may be present without producing symptoms; occasionally diarrhea and nervous symptoms occur.

Diagnosis.—This can be made from the peculiar ova. Living worms are rare in the stool.

Treatment.—Thymol, as previously described. Extract of male fern may be employed. High enemata of warm water, 1 quart (liter), containing 5 to 10 drops of benzin, may be of service.

Trichina Spiralis (Trichiniasis).—The trichina in its adult condition lives in the small intestine. The embryos pass from the intestines and reach the voluntary muscles, where they become encapsulated larvæ.

Muscle Trichinæ.—Tiedemann, in 1822, described the ovoid cysts in human muscle. Owen named the parasite. Leidy, in 1845, described it in the pig. In 1860 Zenker discovered in a girl both the intestinal and muscle forms, and established their connection with the specific symptoms.

Incidence.—Man is infected by eating the raw or not completely cooked flesh of trichinous hogs, which contain encapsulated trichinæ. The capsules are digested in the stomach and the trichinæ set free. They pass into the small intestine, and about the third day become sexually mature. On the sixth or seventh day the embryos are fully developed. The young produced by each trichina (female) have been estimated at several hundred. The female worm penetrates the intestinal wall and the embryos are probably discharged directly into the lymph-spaces, and thence into the venous system, whence they reach the muscles; and in about two weeks they develop into the full-grown muscle form. A myositis is produced and they may become encapsulated. The trichinæ may live therein for many years. Calcification may occur about them. One must remember that in the hog the capsule does not readily become calcified, so that the parasites are not as readily visible as in man. Moreover, an apparently healthy looking animal may be suffering from trichiniasis.

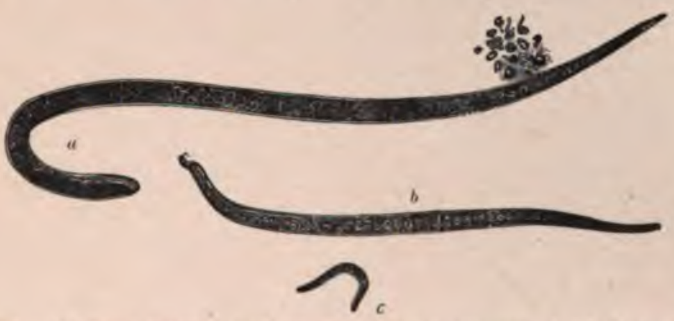

Fig. 268.—Trichina spiralis (greatly enlarged): *a*, Female; *b*, male; *c*, embryo (after Heller).

The intestinal trichinæ are visible to the naked eye—white glistening worms 3 to 4 mm. long; and the male half this size, with two little projections from the hind end. The caudal extremity is thicker than the head (Fig. 268).

The muscle trichina is 0.6 to 1 mm. long and coiled in the capsule. It has a pointed head and rounded tail (Fig. 269).

Symptoms.—As a rule, a few days after eating the infected flesh gastro-intestinal disturbances occur, such as pain in the abdomen, anorexia, vomiting, and at times diarrhea. The attack may resemble cholera nostras or even typhoid fever. Invasion symptoms usually occur between the seventh and tenth days or, occasionally, not until the end of two weeks. There may be fever, intermittent or remittent, even to 102° to 104° F. Chills are not common.

Pain occurs in the muscles on pressure and movement, also swelling of the muscles. There may be difficulty in chewing and swallowing. Dyspnea may be present from involvement of the intercostal muscles and diaphragm. *Edema*, especially *about the eyes*, is an important symptom. Sweating, itching, and urticaria occur. The general nutrition is disturbed and the patient becomes anemic and emaciated. In cases with severe infection, there may be delirium, tremor, and dry tongue, suggestive of typhoid fever. Bronchitis, pleurisy, pneumonia, polyuria, or albuminuria may occur.

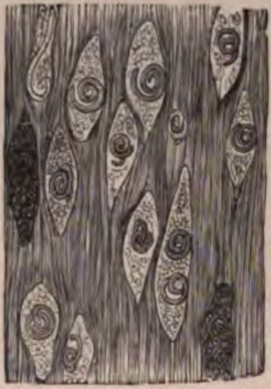

Fig. 269.—Fresh muscle trichinæ (Mosler and Peiper).

Leukocytosis, *especially marked eosinophilia*, is diagnostic.

Eosinophilia, edema of the eyelids, dyspnea, swelling, and tension of the muscles are at once suggestive. The presence of trichinæ in the stools and muscles is conclusive. The muscle can be incised under cocain injection and the cyst examined.

The disease has proved fatal in a number of cases.

Prognosis.—This depends on the *intensity of the infection*. Mild cases may recover in two weeks. The mortality has ranged as high as 30 per cent. Early diarrhea is favorable to evacuate infected pork.

Prophylaxis.—Pork, such as ham, sausage, etc., should always be thoroughly cooked before eating. Proper inspection of hogs is important.

Treatment.—Immediate lavage, if infected pork is suspected and the case is seen early. Evacuation of the bowel by calomel, gr. x (0.6), or a saline cathartic. Thymol, santonin, male fern, kamala, and turpentine, have all been recommended as vermifuges in the early stage. Glycerin in large doses internally is said to be destructive of the worm. Later, the treatment is symptomatic and the strength is to be supported. Urotropin, in gr. x (0.6) doses four times a day, I believe of possible value during the early stages. It is preferable to combine it with equal doses of sodium benzoate.

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